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POPULAR MEDICAL FALLACIES.[1]

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ON being asked to read a paper I found it difficult to decide on any subject, as so much attention has been given of late years to pulmonary tuberculosis. There is hardly any disease in medicine about which more has been written in the form of books and articles on its course, pathology and treatment. This being so, it makes me feel reticent in attempting to give anything pertaining to this subject which will be novel, instructive and interesting.

The title of this paper may to a certain extent be misleading as I will confine my remarks only to the common mistakes in relation to pulmonary tuberculosis.

Climate.

When a person complains of the early symptoms and unhappily very often when he is very ill, the advice frequently given is to "go out west, young man, get a nice easy job in the open and you will do well." Only a few days ago two such arrived here on advice given them by medical men in Collins Street, Melbourne. One of them had a few shillings and the other, a married man with a wife and family, had only £15 to bless himself with. It is almost needless to say that man cannot live on air alone and it must be clearly understood that "nice easy jobs" in the open places of the Queensland bush hardly exist. There are too many able bodied men looking for work to allow soft billets to remain idle and they take them eagerly, until something better turns up. If the sick one be lucky enough to get one of these light easy jobs, he will soon discover it also means a light easy pay, earned under the most unhygienic conditions with rations consisting of dry corned beef and badly cooked damper which can be appreciated only by strong, healthy men with good appetites. Even with a few pounds troubles unthought of will be encountered from unsympathetic publicans and cheap boarding house keepers. If he should be fortunate enough to meet some kind sympathetic soul and there are few, the man already quoted will find that his £15 will not carry him very far. Kindly disposed lodging house

[1] Read at a meeting of the Queensland Branch of the British Medical Association on September 4, 1925.

keepers have to live. This being so, the patient would have done better for himself and his health by resting in his own back yard.

A good climate is very difficult to define. To me a climate is a good one where a patient can pass all his time both day and night breathing pure air with the greatest amount of comfort. This may be found in the highlands and plateaux of Australia. Moist heat or moist cold air, besides being deleterious, is intensely disagreeable to a lung-sick man. The seaside is almost universally condemned in Australia. I could never exactly make out why; Cannes, Nice, the Italian Riviera, Canary Islands and Madeira are justly celebrated. The only disadvantage is the crowded casinos and dancing halls often frequented by invalids. In winter the climate of the seaside resorts in Queensland is ideal, nothing better can be found in the world.

In a book recently written by Pottinger—the best he has ever produced—on climate and advice to patients he sums up the question of climate in a concise sentence. "I would rather be treated in a bad climate under careful and scientific control than run wild in the best."

I will not criticise Leonard Hill's epoch making researches on pure and stagnant air, as they are beyond the scope of this paper, but will content myself by pointing out that all his experiments were made on strong, lusty students and will conclude by quoting Halliday Sutherland in his criticism of Hill's investigations.

> Air may be cooled, washed, sterilized and washed again. Chemically it is pure and may contain the same gaseous constituents as untreated air. Those who breathe it find something lacking—the air is dead. Pure water in a tank has not the tonic qualities of a mountain stream.

Housing and Poverty.

At the present time a great deal is being spoken and written, especially by sanitarians, about poverty and its natural concomitant bad housing. There is the usual juggling with figures when calories and consumption are proved to be exactly in the inverse ratio, that is to say, low calories indicate increased pulmonary tuberculosis. The cry is: "Give every one good nourishing food and house him well and phthisis, the bane of existence, will be no more, as this disease is the result of malnutrition and bad environment." Like David Harum, I am not the seventh son of a seventh son, nor was I born with a caul, still in spite of these disqualifications I venture to prophesy that if it were possible by one fell swoop to wipe off poverty and all the slums existing in this the best of all possible worlds, one would never get rid of tuberculosis by this means alone. I do not wish to assume the rôle of a captious critic of this proposed latter day campaign, if you can call it such, but as far as I have read, it seems to me that the tubercle bacillus has been forgotten. To quote the well known phrase of Professor Glaister: "Broken down houses mean broken down occupants" and resulting malnutrition goes hand in hand with this bad environment, so as to aid the progress not only of tuberculosis, but also of every other infectious disease. Before finishing the consideration of this I will go further by saying that taking it for granted that treatment and after care are in every way equal, the slum dweller afflicted by this disease makes a quicker and more satisfactory recovery than do folk in good circumstances.

Occupation.

Relatively the mortality in this disease is high in publicans, barmaids, painters and shoemakers and in this connexion much has been said and written about bad habits and alleged causes. The truth is, the real cause is infectious dirt. Though in sympathy with prohibition, I am sorry to confess I think that the prohibitionists have indulged in a lot of superfluous talk against the use and abuse of alcohol. They have lost sight of the fact that those engaged in the "trade" are exposed to more infection than other people. As I mentioned under the consideration of housing, neglect or abuse of any kind is conducive to all infective disease.

The affected man feels tired and life does not look too rosy. He enters the public house for a "spot" and ends in having half a dozen or more. With each drink he loses his usual caution and promiscuous spitting is the usual accompaniment of the pipe or cigarette.

Some unwarranted talk has been made of the dangers accruing from the noxious fumes of fresh paint as being injurious to the lungs. The fact is that a painter's job is a dusty one and means peeling off old papers or the scraping of dirty and sometimes infected walls before he can get a good foundation to start his real work. The same applies to shoemakers. Repairing shoes is a dirty trade and is a highly probable means of infection. Certainly it is a more potent factor than posture which generally bears the blame. The same may be said of laundresses whose death rate is high. Infection from dirty linen and soiled handkerchiefs is of more importance than the continual wet and general discomfort of washing.

Hæmorrhage.

There are more fallacies about hæmorrhage than any I have yet mentioned.

Hæmorrhage may also occur in other diseases such as hydatids, spongy gums, gastric ulcer and so forth, but these as a rule can be easily diagnosed. In mitral stenosis there is sometimes considerable hæmorrhage which is misleading even to the astute practitioner. In such with the bleeding there may be cough, sputum, impaired percussion note, bronchial breathing and râles, varying in quality and heard in the right upper lobe. In fact, the *tout ensemble* cannot be distinguished from pulmonary tuberculosis of the right lung. I now confidently affirm that these conditions are non-tuberculous affections of the lung, notwithstanding the diagnosis of the radiologist. This was a favourite theme of the late Professor Grainger Stewart who held that mitral stenosis and tuberculosis never existed concurrently and he refused even in rare cases to admit the possibility, unless tubercle bacilli were demon-

strated in the sputum. The explanation of the X ray plate is easy. What was presumed to be tuberculous deposit by the radiologist, is really a relic of former simple inflammation in the pulmonary lobules. It is a very good example of what Sir Thomas Horder terms the "bastard pathology" of the radiologists.

All the diseases noted above being excluded, hæmorrhage from lungs is due to one cause only—tuberculosis and is always a sign of active disease with one exception, namely rupture of an aneurysm of the pulmonary artery in a healed cavity.

Exertion as a cause of bleeding is a common fallacy. The truth is that exertion is bad for any symptom of this disease. The very worst cases occur in patients who have been in bed for months. Ice bags, cold drinks, swallowing chips of ice and a low diet consisting of milk is a superstition deeply ground into the minds of the profession and the laity, almost amounting to a fetish. The poor soul is miserable and apprehensive enough without making him worse with cold applications. Swallowing chips of ice in gastric ulcer I can readily understand, but in bleeding from the lungs what good can it do? Depletion with low diet, when the patient has already lost enough by a large hæmorrhage, is bad practice. Warm, nicely cooked and easily digested food is what he often craves for. Why not let him have it? Unfortunately the laity is so impressed with the idea that sparse diet is necessary, that they think the doctor is not treating the patient properly if he does not order it. Giving milk alone is as bad as treating enteric fever in the same way.

Infection and Disease.

At present the impression in the minds of many seems to be somewhat vague in differentiating infection from disease. I admit that the borderland in the obscure light of our knowledge up to date is difficult to define; this I put down chiefly to the malinterpretation of the radiogram or to put it more explicitly the malinterpretation by the practitioner of the interpretation of the radiographer. To begin with I am not a radiologist, therefore I approach this subject with timidity, still, though running risk of being charged with undue obstrusiveness, I venture to remark that the whole subject seems to me to be in the melting pot and that the X ray man allows his impressions to run away with him; also he has not realised that infectivity does not necessarily imply infectiousness. I have been told on fairly good authority that one eminent radiologist in Melbourne refrains from reporting old lesions or anything else that does not actually pertain to the diagnosis of an active area. If this be so, I take off my hat to him in the profoundest respect. I think that when the lesion on the plate appears to be doubtful, it is only fair to give the patient the benefit of the doubt. In nearly every case in which there is activity in the apex of one lung, there is evidence of latent disease in the apex of the other; this is found on the X ray plate. In other words pulmonary tuberculosis is nearly always bilateral, one side being as a rule more advanced than the other. For instance when there is clinical evidence that one apex is affected, to tell the patient there is slight evidence of infectivity in the other is the height of cruelty. The only conclusion the laity can draw from such a statement is that both lungs are diseased. The psychic element produced is so pronounced that it has often led to mental aberration. In a paper lately published by Lynham, Radiologist to the Mount Vernon Hospital, he states that the impressions given by the screen are superior to the film.

> One can watch the expansion and contraction of the chest, or of any given part of the lung and detect degrees of lighting up or defective translucence which are often lost in the film. A screen examination gives incomparably better information than a bad film. Also one can move the patient about and get a series of views at the one sitting.

He then goes on to say:

> Now there is nothing pathognomonic in such shadows. I do not think it is ever possible to be absolutely positive from a radiogram alone, that a patient has tuberculosis. It is possible that other diseases give rise to similar shadows. So I make it a rule never to say "this is tuberculosis," but rather say, "these shadows suggest tuberculosis."

With increasing knowledge and experience many difficulties that exist at present will be cleared away and I look forward with great hope to the time when X ray work in the chest troubles will be one of the surest aids in making a diagnosis.

Elsewhere I drew attention to the apparent neglect of radiologists concerning the normal chest. In a paper lately published by Clive Rivière on the "Normal Chest X-rayed" the pitfalls of radiology are described.

Mechanism of the Respiratory Murmur.

In 1878 Bordet and Cheauveau published their studies on the mechanism of the vesicular murmur. In a horse suffering from pneumonia of the left lung tracheotomy was performed. On wide separation of the tracheal wound with retractors while the chest was auscultated, all the respiratory sounds disappeared in the affected lung.

Colonel Bushnell, head of the Medical Department of the United States Army stationed in Washington, revived this study, looking at it from a different angle; he confined his observations to the movements of the larynx and simultaneous auscultation of the chest.

When the larynx opens during an inspiration the cords relax and this is caused by the contractions of the posterior crico-arytenoid muscles which rotate the arytenoids. During expiration these muscles relax and the cords pass into their position of rest. Bushnell conceived the idea that if the glottis could be kept open (assuming that the sound of the so-called vesicular murmur originated in the larynx) the murmur would not be heard. In other words a silent larynx means a silent lung. Bushnell found that after considerable practice he could control the action of his laryngeal muscles sufficiently during expiration and inspiration as to be able to submit himself to a test before witnesses

and for this reason two were chosen, one a laryngologist of repute and the other a physician. During the test it was noted that the respiratory murmur disappeared. This is certainly interesting, but I would like to hear it confirmed in Australia. On reading Bushnell's paper, try as I might, I could not divest myself of the feeling that the article was written with a certain amount of bias. The vagaries of preconceived opinions in men of the highest rectitude are very well known to psychologists, so I feel absolved from any shadow of a suspicion that I impute any sinister motives on the part of Colonel Bushnell who commands the greatest respect amongst his colleagues. Notwithstanding this, the feeling exists in my mind. I think it was Pasteur who said it is surprising what a man whose honour is above reproach, will see through the bore of a microscope if he expects to see it.

The man who has complete control of his muscles, is difficult to find; the only man in Australia who possesses this qualification I am told, is Dr. Royle of Sydney.

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BACILLARY DYSENTERY IN INFANCY AND CHILDHOOD.

Part I.

Dysentery Bacilli as Recovered from the Motions of Infants and Young Children.

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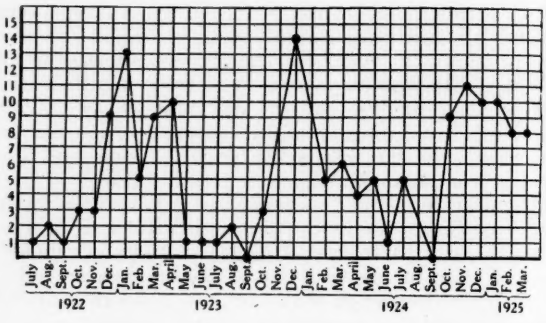
Introductory.

Among the complexity of ætiological factors which operate in the diarrhœal disorders of children and particularly infants, one agent, the dysentery bacillus, stands alone. Particularly ominous by reason of its death dealing capacity, *Bacillus dysenteriæ* is responsible for a specific infectious disease the identity of which is apt to be lost under the loose nomenclature, "summer diarrhœa."

The bacillary dysentery of infancy and childhood is not a disease of the summer months exclusively. Although it assumes epidemic proportions at this time, infantile bacillary dysentery, as experienced at the Children's Hospital, Melbourne, occurs in every month of the year. The following chart (Table I.) in which is graphically recorded the number of individual children from whom *Bacillus dysenteriæ* was recovered in successive months from July 1, 1922, to March 31, 1925, is drawn not to labour the truism that bacillary dysentery is especially prevalent in the summer months, but to show that its incidence is by no means restricted to the warm season.

Table I.



To the discriminating clinician the term "summer diarrhœa" conveys nothing. He regards it as nothing more than a symptomatic description of illness which may be the expression of dietetic mismanagement on the one hand or acute and serious infection on the other.

Bacteriological.

It is proposed to consider one hundred and fifty-three strains of dysentery bacilli recovered from the stools of infants and young children during the period July 1, 1922, to March 31, 1925 and the classification of one hundred and twenty-one of them into serological types.

In 1922-1923 one of us (F.E.W.), in association with Dr. S. W. Patterson, was engaged at the Walter and Eliza Hall Institute in work relating to the serological classification of dysentery bacilli as they occur in Australia and included among the strains examined at that time were thirty-seven isolated from the motions of infants at the Children's Hospital and forwarded to the Institute. The findings in respect to these thirty-seven strains are embodied in the following discussion. As it seemed of importance from the point of view of serum therapy to obtain more extended observations on the serological races of dysentery bacilli peculiar to infantile diarrhœa in this State the work was carried on at the Children's Hospital during 1923, 1924 and the early part of 1925 with the object of determining:

1. The incidence of the several races;
2. Possible seasonal variations in the prevailing races;
3. The comparative virulence and attendant mortality in the different serological groups.

The monovalent agglutinating sera requisite for the identification of the several serological types

were prepared by the immunization of rabbits with standard strains representative of the five races recognized among the Flexner or mannite-fermenting dysentery bacilli.

No attempt will be made to record the details of the diversity of other microorganisms encountered in the search for dysentery bacilli. A bewildering multiplicity of bacteria besets every worker in this field and the complexity of the bacteriological findings in infantile diarrhœa is seen in the careful work of Forsyth.[1] The volume of routine bacteriological examinations of infants' stools was such as to preclude any possibility of reaching finality regarding the pathogenic significance of the varied assortment of bacteria encountered. Certain of them, however, on account of their resemblance to *Bacillus dysenteriæ* or other special features will be discussed.

Bacillus Dysenteriæ.

Under the generic term of *Bacillus dysenteriæ* are included a large number of microorganisms which received intensive study during the great war. They may be said to fall into two main groups or species according to their capacity to ferment mannite. On the one hand the non-fermenters of mannite included the Shiga bacillus and Schmitz's bacillus, which differs from *Bacillus dysenteriæ* (Shiga) in possessing the property of elaborating indol in appropriate media. The fermenters of mannite include a variety of microorganisms embraced in the general description *Bacillus dysenteriæ* (Flexner).

As between the Shiga bacillus and the Flexner group the Shiga is the more dangerous. It may be said to be a specialized and homogeneous type the members of which exhibit no differentiation into serological groups.

The Flexner group on the other hand comprises a number of races serologically distinct and also includes various slow fermenters of lactose which are now recognized as dysentery bacilli in the sense that they are capable of producing clinical dysentery.

The following table, modified from Andrewes and Inman[2] illustrates the principal differences between the various dysentery bacilli and the manner in which the mannite fermenting group is subdivided according to the action of its members on lactose and dulcite.

Although divisions among the mannite-fermenting dysentery bacilli became apparent by the investigation of their sugar fermenting properties, it is now accepted that the fermentative powers afford an unstable basis of classification on account of their liability to variation. Serological methods in the hands of Kruse,[3] Murray[4] and finally of Andrewes and Inman[2] elucidated five component groups among the Flexner dysentery bacilli. These have been designated by the last five letters of the alphabet, *V, W, X, Y* and *Z* and there are indications that sub-groups exist, such as a sub-group of *V* with a pronounced *Z* element in its composition (*VZ* group).

The Shiga bacillus may be said to be a negligible factor in infantile bacillary dysentery in Melbourne. All the strains isolated in the period under review belonged to the mannite-fermenting Flexner group, there being included among them nine examples of the Sonne bacillus.

The Sonne Bacillus.

Among the mannite-fermenting non-gas-producing slow fermenters of lactose the bacillus first described by Sonne is constituted an entity by its special serological characters. Unless a rigid routine is pursued in the investigation of suspected Flexner bacilli, the Sonne bacillus may escape identification as up to a point it gives identical reactions in sugar containing media and is even agglutinable in low titre by Flexner sera. Its special features are the production of acid in lactose, discernible by means of a sensitive indicator after a period of incubation varying from seven to twelve days and specific agglutinogenic properties.

It had been shown (Patterson and Williams[5]) that by immunization of rabbits with a strain of the Sonne bacillus a serum is obtained which, while agglutinating homologous strains in very high titre (1:6,000), affects representatives of the *V, W, X, Y* and *Z* races of Flexner bacilli either in very low titre or not at all. Conversely, the only monovalent

TABLE II.—SUBDIVISION OF MANNITE FERMENTING GROUP OF ORGANISMS.

Organism.	Sugars in the Medium.				Indol.	Agglutinating Sera.					
	Glucose.	Mannite.	Lactose.	Dulcite.		Shiga Serum.	Schmitz Serum.	Flexner Serum In Variety.	Bacillus Alkalescens	Lactose Fermenters in Variety.	Gettings's Bacillus.
Shiga's bacillus	A	—	—	—	—	+	—	—	—	—	—
Schmitz's bacillus	A	—	—	—	p	—	±	—	—	—	—
Flexner's bacillus	A	A	—	—	tr	—	—	+	—	—	—
Bacillus alkalescens	A	A	—	A	p	—	—	—	+	—	—
Lactose fermenters in variety	A	A	A	A	tr	—	—	—	—	+	—
Gettings's bacillus	A	A	—	—	p	—	±	—	—	—	+

A = acid formation; p = present; tr = trace present; + = agglutination; ± = feeble agglutination; — = no agglutination.

serum of the Flexner races capable of affecting the Sonne bacillus is that of *X*, but agglutination does not proceed to high titre. The workers named further demonstrated that heavy absorption of *X* serum with Sonne bacilli leaves the specific agglutinin for *X* untouched.

The Isolation of Dysentery Bacilli and Identification of Serological Race.

It is not proposed to describe the routine pursued in the isolation of dysentery bacilli in detail. It may be indicated that the MacConkey medium was used throughout for the preliminary plates, that motility and the action of microorganisms from likely non-fermenting colonies on lactose, glucose, mannite, sorbite, dulcite and cane-sugar were examined in appropriate media and identification established by agglutination tests with the polyvalent antidysenteric serum issued by the Commonwealth Serum Laboratories. Some strains agglutinated with this serum in titre of 1:3,200, the majority at 1:1,600, but an agglutination titre of 1:800 was accepted.

In the next instance agglutinable emulsions, designed for the work of classifying the strains with reference to serological race were prepared from those cultures identified as Flexner dysentery bacilli. On account of the fact that the susceptibility to agglutination by a specific serum of a given bacterial emulsion is affected by the period of subculture of the organism and the strength of the emulsion, an endeavour was made to secure uniformity as regards these conditions throughout the series. With this object the emulsions for agglutination of all the strains to be examined were prepared from the same generation of subculture, that is, the third and approximate standardization of the strength of the emulsions was secured by making each with ten cubic centimetres of saline solution and the bacteria to be obtained from a well covered agar slope after eighteen hours' incubation. Such bacterial emulsions were preserved by the addition of 0.5% "Formalin."

The agglutination tests were performed by the macroscopic technique, the tubes containing the serial dilutions of serum and bacteria being incubated at 37° for four hours and allowed to stand overnight before readings were taken. In the tables which follow a single *plus* sign (+) is used to

TABLE III.—TITRE OF AGGLUTINATING SERA FOR HOMOLOGOUS AND HETEROLOGOUS RACES.

Sera.	Emulsions of Bacilli.					
	V.	*W.*	*X.*	*Y.*	*Z.*	Sonne.
V (Elstree)	1 : 8,000	1 : 100	1 : 800	1 : 800	1 : 800	1 : 200
W (Cable)	1 : 400	1 : 3,200	1 : 100	1 : 400	1 : 100	(—)
X (Hughes)	1 : 800	1 : 200	1 : 3,200	1 : 800	1 : 800	(—)
Y (His and Russell)	1 : 400	(—)	1 : 1,600	1 : 8,000	1 : 3,200	1 : 400
Z (Whittington)	1 : 800	1 : 100	1 : 100	1 : 200	1 : 3,200	1 : 400
Sonne	(—)	(—)	(—)	(—)	(—)	1 : 6,400

(—) = less than 1 in 100.

TABLE IV.—RESULTS OF AGGLUTINATION TESTS WITH ELEVEN ILLUSTRATIVE STRAINS.

Culture.	Maximum Titre of Serum for the Standard Strains.				
	V (8,000).	*W* (3,200).	*Y* (8,000).	*Z* (3,200).	Sonne. (6,400).
69	1:500 (—)	1:400 (++) 1:800 (+)	1:500 (—)	1:200 (—)	1:200(—)
81	1:500 (++)	1:800 (++) 1:1,600(+)	1:500 (—)	1:200 (—)	1:200(—)
94	1:400 (++)	1:400 (+)	1:500 (—)	1:400 (++)	1:200(—)
77	1:500 (—)	1:200 (—)	1:500 (—)	1:400 (++) 1:800 (+)	1:200(—)
72	1:4,000(++) 1:8,000(+)	1:200 (—)	1:500 (—)	1:400 (++) 1:800 (+)	1:200(—)
87	1:2,000(++) 1:4,000(+)	1:200 (—)	1:500 (—)	1:400 (++) 1:800 (+)	1:200(—)
84	1:500 (—)	1:400 (++) 1:800 (+)	1:500 (—)	1:800 (++)	1:200(—)
89	1:1,000(++)	1:400 (++)	1:8,000(++)	1:200 (+)	1:200(—)
67	1:4,000(++) 1:8,000(+)	1:200 (+)	1:500 (—)	1:400 (+)	1:200(—)
85	1:500 (—)	1:200 (—)	1:500 (—)	1:400 (++) 1:800 (+)	1:200(—)
48	1:500 (+)	1:1,600(++)	1:500 (—)	1:200 (+)	1:200(—)

indicate flocculation visible without the aid of a lens but short of clarification of the saline solution. Triple *plus* (+++) and double *plus* (++) signs indicate that clarification was complete, the former being employed when it was effected within the four hours of incubation.

The specific agglutinating sera for the *V*, *W*, *X*, *Y* and *Z* races of Flexner group dysentery bacilli and for the Sonne bacillus, prepared as already indicated by the immunization of rabbits, exhibited maximum titres for homologous and heterologous races as shown in Table III.

As a preliminary step in the identification of serological races twenty-four strains were examined with regard to their behaviour when subjected to agglutination by the above type sera and indications of the various races into which they fell, gained by noting that serum with which the maximum titre was registered or with which agglutination was carried to a titre beyond the zone of group agglutination. Space does not allow of the reproduction of full details of the agglutination tests, but in Table IV. are assembled eleven illustrative strains. Among these are some in which the serological race is apparently clearly indicated, but in the case of others there is so much cross-agglutination that no conclusion can be formed.

In the above table are three strains, No. 69 (*W*), 77 (*Z*) and 85 (*Z*) which did not exhibit cross-agglutination in the dilutions of the sera employed, but in the other seven and in the majority of the twenty-four subjected to this preliminary examination group agglutinins operated to cloud the clear definition of race.

Absorption of Agglutinin: Principle.

Biologically and morphologically similar bacteria, such as those representing the several races of dysentery bacilli, might be expected to have much in common as far as their agglutinogen functions are concerned. That an agglutinating serum prepared from any one of them will contain major agglutinins for the homologous race and minor agglutinins for allied races is to be anticipated in accordance with the principles governing group agglutination.

Table III. shows the extent to which minor agglutinins were present in the sera at our disposal and in 94 and 89 of Table IV. the confusing effect of the minor or group agglutinins is illustrated.

To clarify the results and dispense with cross-agglutination as far as possible the absorption of agglutinin technique was applied after the examination of the first twenty-four strains. After revision of these strains by agglutination tests with "absorbed" sera, the work was thenceforward carried on with sera in which the minor agglutinins were reduced to a point at which they were ineffective in producing cross-agglutination.

It was first shown by Castellani that absorption of a serum with the organism used for immunization removes not only the major agglutinins but all of the agglutinins, major and minor. Conversely, absorption of such a serum with the species agglutinated by the minor agglutinins takes out these antibodies only, leaving the major agglutinins intact.

On this principle each of the four sera *V*, *W*, *Y* and *Z* was treated with heavy emulsions of the bacteria of the other three races with a view to removing the minor agglutinins from each. Thus the *W* serum as prepared, although possessed of a titre of 1:3,200 for the homologous *W* (Cable) strain also agglutinated the *V*, *Y* and *Z* type strains in maximum dilutions of 1:400, 1:400 and 1:100 respectively. By treating the *W* serum with heavy emulsions of *V*, *Y* and *Z* cultures it was rendered ineffective in agglutinating these races in the low dilution of 1:100, while its capacity to agglutinate its homologous *W* race was only slightly reduced.

In this manner each of the several type sera was rendered for practical purposes specifically monovalent.

Technique: It was not the objective to reduce the minor agglutinins to vanishing point; indeed it is doubtful if it is possible to exhaust a serum completely of its agglutinins. It was found that an absorbing dose of 100,000 million organisms per cubic centimetre of undiluted serum was sufficient to eliminate the minor agglutinins as confusing factors. The following procedure was adopted. Heavy emulsions of the organisms to be employed for absorption of agglutinin were prepared by passing five cubic centimetres of saline solution over each of ten agar slant cultures in succession, so that at the end of the series rather less than five cubic centimetres of a very dense emulsion were collected. Portion of this was diluted for counting and the number of bacilli per cubic centimetre enumerated in a hæmocytometer chamber, the counting not being commenced until the organisms had been allowed to "settle" for half an hour. In one series of absorption procedures which may be given by way of example, emulsions of the following strengths were prepared:

V (Elstree) .. .. 40,000 million per cubic centimetre
W (Cable) .. .. 40,000 million per cubic centimetre
Y (His and Russell) 36,000 million per cubic centimetre
Z (Whittington) .. 25,000 million per cubic centimetre

To one cubic centimetre of undiluted *W* serum were added:

2.5 cubic centimetres of *V* emulsion (100,000 million bacteria).
4 cubic centimetres of *Z* emulsion (100,000 million bacteria).
2.5 cubic centimetres of *Y* emulsion (90,000 million bacteria).

The *W* serum was thus exposed to absorption of its agglutinins for the *V*, *Y* and *Z* races in dilution of 1:10. After incubation for four hours the absorbing mixture was allowed to stand in the ice-chest over night. With the aid of centrifuging on the following morning clear serum in dilution of 1:10 was obtained.

The other three sera were similarly treated. Each was exposed to approximately 100,000 millions of each of the three heterologous races per cubic centi-

metre of undiluted serum. The agglutinating properties of the sera after absorption are shown in the following table.

The results obtained by agglutination tests with the sera subjected to absorption of agglutinin and the unequivocal declaration of race they embodied may be illustrated by consideration of some of the strains in Table IV. Reference to this table will show that 81, for example, when tested against untreated sera exhibited agglutination (++) with the *V* serum in dilution of 1:500 and with the *W* serum (+) in dilution of 1:1,600. The presumption was that this was a member of the *W* race affected by the *V* serum by virtue of its *W* group agglutinin content. That this was so was established by the fact that when sera in which the minor agglutinins were reduced, were employed, this strain was no longer susceptible to agglutination by the *V* serum, but reacted as before to the *W* serum.

Number 72 provides an example of cross-agglutination between the *V* and *Z* sera. That the strain belonged to the *V* race became clear when it transpired that its agglutination by the *Z* serum was effected by a group agglutinin. The *Z* serum after absorption by bacteria of the *V* race was no longer capable of agglutinating this strain in the lowest dilution in which it was employed (1:200), but the strain still reacted well with the *V* serum subjected to absorption by bacilli of the *Z* race.

Considerations of space render it impossible to reproduce all the tables of agglutination tests and the titres registered with the several sera before and after absorption by the one hundred and twenty-one strains. Sufficient detail has perhaps been given to indicate the principle on which the work of classifying the strains with reference to serological race was carried out.

The behaviour of certain of the strains, of which 94, 72, 87 and 67 in Table IV. are representatives, merits further discussion. From their agglutination reactions as set out in the table it appeared that these strains belonged to the *V* race, but were at the same time susceptible to a degree of agglutination by the *Z* serum. The reaction with *Z*, however, persisted in the case of all four after the *Z* serum had been exposed to an absorbing dose of 100,000 million *V* bacteria per cubic centimetre. Even after reabsorption of the *Z* serum with a further 100,000 million *V* organisms the strains under consideration were still affected by the *Z* serum in dilution of 1:200. It appeared impossible to reduce further the agglutinins in the *Z* serum for these strains which at the same time were agglutinable in high titre by the *V* serum.

The majority of the *V* strains encountered were not thus susceptible to agglutination by the *Z* serum after its absorption by *V* bacteria and it is suggested that those which behaved in this manner, possess an antigenic structure in which, although the *V* antigen is dominant, *Z* is at the same time strongly represented—*VZ* group.

Of the twenty-nine strains identified as of the *V* race eight would perhaps be more properly described as belonging to a sub-group *VZ*.

It will be noted that no *X* serum figures in the agglutination tests comprising Table IV. or in the outline of absorption procedures adopted.

The thirty-seven cultures of dysentery bacilli recovered from infants and classified during 1922-1923 contributed to make a series of two hundred strains gathered from all sources in Melbourne and Sydney and investigated at the Walter and Eliza Hall Institute. In these two hundred strains the *X* race was not represented by a single member. Although there were many strains susceptible to a monovalent *X* serum in varying titres, none of them was affected by the same serum in the low dilution of 1:100 after it had been exposed to absorption by emulsions of bacteria of the heterologous races, *V, W, Y* and *Z*. On the other hand such strains were quite satisfactorily and specifically agglutinated by one or other of the *V, W, Y* or *Z* sera from which the agglutinins for heterologous races had been removed by absorption.

In view of this experience the *X* serum was not included in the routine agglutination tests applied to the organisms isolated in the second and third seasons here reviewed and it was found that with the exception of four strains all those investigated could be placed by a specific agglutination reaction with the corresponding serum in the *V, W, Y* or *Z* group. It was judged that should any *X* strains occur, an indication of their identity would be afforded by a degree of agglutination occurring with all the other four sera, as no steps were taken to remove the *X* agglutinin from them by absorption. The only cross-agglutination observed, however, when absorbed sera were employed, was that occurring between the *V* and *Z* groups as already described.

TABLE V.—REACTIONS OF *V, W, Y* AND *Z* SERA WITH HOMOLOGOUS AND HETEROLOGOUS RACES AFTER REMOVAL OF MINOR AGGLUTININS BY "ABSORPTION."

Sera.	Emulsions of Bacilli.			
	V.	*W.*	*Y.*	*Z.*
V (Elstree)	1:4,000(++)	1:500 (—)	1:500 (—)	1:500 (—)
W (Cable)	1:100 (—)	1:1,600(++)	1:100 (—)	1:100 (—)
Y (His and Russell)	1:500 (—)	1:500 (—)	1:8,000(++)	1:500 (—)
Z (Whittington)	1:100 (—)	1:100 (—)	1:100 (—)	1:1,600(++)

It has been indicated that four strains were refractory in that they were not agglutinable to satisfactory titre with the absorbed sera *V*, *W*, *Y* or *Z* or with the serum prepared against the Sonne bacillus. These four were assembled and examined as regards agglutinability by the *X* serum. Low titres were recorded before the minor agglutinins were removed from the serum, but its titre for each of the four organisms was reduced to less than 1:100 by exposure to absorption of agglutinin with heavy emulsions of the other races.

Incidence of the Several Races.

In Table VI. will be seen the extent to which the individual serological races were represented among dysentery bacilli isolated from the motions of young children during three successive seasons.

It will be seen from Table VI. that the *W* race was predominant in the first and third years under review and that in the second year it divided pride of place with the *V*. The important facts emerge that the two races *W* and *V* embraced eighty-five or 70.2% of the one hundred and twenty-one strains which were examined with respect to serological type and that the W strains alone represented 46.2%.

The nine examples of the Sonne bacillus figured as occurring in the first of the three seasons were identified as such at the Walter and Eliza Hall Institute (F.E.W.).

The chronological occurrence of these representatives of the Sonne bacillus is of some interest. The infants from whom they were isolated were all admitted to the Children's Hospital between December 19, 1922, and March 8, 1923, and during that period dysentery bacilli as recovered from seventeen children were classified. No less than nine of the seventeen were Sonne bacilli and five of the Sonne strains were recovered consecutively from the last five children identified as subjects of bacillary dysentery for the period indicated. There would thus appear to have been a "wave" of Sonne infection at this time and it is remarkable that among the eighty-four strains of dysentery bacilli classified since that date, no further examples of the Sonne bacillus have been recognized. It is unlikely that any have been overlooked for the following reasons.

Every strain examined with respect to serological race was subjected to agglutination with a high titre (1:6,400) serum specific against Sonne bacilli. In none of the strains investigated was there any agglutination with this serum even when it was used in as low a dilution as 1:200. Further, all the strains were agglutinable to satisfactory titre by one or other of the *V*, *W*, or *Z* sera, the removal of group agglutinins from which by absorption had rendered them specific for the *V*, *W*, *Y* and *Z* races respectively.

Some interesting material was provided by an outbreak of bacillary dysentery which occurred among the infants of the Children's Welfare Department at Royal Park in July, 1924. It may be noted in passing that this was a winter epidemic. Seven strains were furnished by these children and as was to be anticipated, all were of the same serological race (*W*). The next subject of bacillary dysentery received from this institution was admitted to the Children's Hospital on October 10, 1924, and the sequence of *W* strains was then broken by the recovery of one of the *Y* race. From that time to the closure of the series here reviewed no further strains were obtained from inmates of the Children's Welfare Department.

In two families in which more than one child was affected, the race of dysentery bacillus was the same in each child. Numbers 42 and 45, both *W* strains, and Numbers 113 and 115, *V* strains, were recovered from brothers and sisters.

The Serological Races Considered with Respect to Comparative Virulence and Mortality.

The following table is constructed to show comparatively the mortality occasioned by the several serological races of *Bacillus dysenteriæ* (Flexner) as they occurred in the three seasons under review.

From this table (Table VII.) it will be seen that there is little to choose between the different races of Flexner bacilli all having been responsible for a death rate in the region of 50% during 1923-1924 and 1922-1923. The mortality figures for all the races fell notably in the season 1924-1925 and it is

TABLE VI.

Race.	1922-1923.		1923-1924.		1924-1925.		1922-1925.	
	Number.	Percentage of Total.	Number.	Percentage of Total.	Number.	Percentage of Total.	Number.	Percentage of Total.
V	5	13.5	14	34.1	10	23.3	29	23.9
W	16	43.2	17	41.4	23	53.3	56	46.2
Y	3	8.1	4	9.7	4	9.3	11	9.0
Z	4	10.8	6	14.6	6	14.0	14	13.2
Sonne	9	24.3	—	—	—	—	—	—
Totals	37		41		43		121	

The 1922-1923 group was investigated by F. Eleanor Williams; the 1923-1924 and 1924-1925 groups by Reginald Webster.

TABLE VII.

Race.	1922-1923.			1923-1924.			1924-1925.		
	Recovered.	Died.	Death. Rate.	Recovered.	Died.	Rate. Death.	Recovered.	Died.	Death. Rate.
V	2	3	60.0%	8	6	42.8%	9	1	10.0%
W	6	11	64.7%	10	7	41.2%	16	6	27.3%
Y	1	2	66.6%	3	1	25.0%	3	1	25.0%
Z	2	2	50.0%	3	3	50.0%	3	3	50.0%
Sonne	4	5	45.5%	—	—	—	—	—	—

In 1922-1923 the general mortality rate among fifty-seven children, including twenty infected with bacilli of which the serological race was not determined, was 56.1%.
In 1923-1924 the general mortality rate among forty-nine children, including eight infected with bacilli of undetermined type, was 42.8%.
In 1924-1925 the general mortality rate among forty-seven children, including five infected with bacilli of undetermined type, was 29.4%.

interesting to compare the general mortality rates in the three seasons. The summer of 1922-1923 was distinguished by very heavy mortality; in that of 1924-1925 the death rate was low for dysenteric infection in infancy and the intervening season occupied an intermediate position in this respect. From Table VII. it would appear that the occurrence of a "bad" season does not depend upon high incidence of infection by any particular race distinguished among its fellows by excessive virulence, but rather upon an access of virulence in which all the races share equally.

Dysentery-like Bacilli.

In Table VIII. are assembled the reactions of a number of microorganisms which are recorded on the ground that they cannot be summarily excluded from the dysentery group. In the case of every bacterium included in the table the infant from whom it was recovered, passed stools of a character highly suggestive of dysenteric infection. All the organisms were non-motile and when they affected the sugars, did so without the production of gas. The majority were non-fermenters of lactose, that is, no indication of acid production from this sugar was apparent after twenty-one days incubation, but Nos. 2 and 3 produced acid in lactose on the third and eighth day of incubation respectively.

Concerning the slow fermenters of lactose it is to be noted that Andrewes and Inman[2] (see Table II.) give a place among dysentery bacilli to "lactose fermenters in variety" and in this category is to be found the Sonne bacillus the pathogenicity of which is now not to be questioned.

Such investigation of the organisms in Table VIII. as a busy daily routine permitted consisted of a determination of the agglutinability of the several bacilli by the sera of the infants from whom they were recovered. In no case did the serum of any child exhibit agglutinating capacity for the homologous organisms even in dilutions as low as 1:20.

TABLE VIII.—DYSENTERY-LIKE BACILLI.

Number.	Motility.	Fermentation Reaction in							Remarks.
		Lactose.	Glucose.	Mannite.	Sorbite.	Dulcite.	Cane-sugar.	Litmus-milk.	
1	—	—	A. 1	—	A. 2	—	—	Alk.	Inagglutinable by Shiga serum; child died.
2	—	A.3	A. 1	A. 1	A. 1	—	A.2	A.&Clot	Inagglutinable by Sonne serum; child recovered.
3	—	A.8	A. 1	A. 1	A. 1 Alk.10	—	—	A.&Clot	Inagglutinable by Sonne serum; child recovered.
4	—	—	A. 1	A. 2	A. 4	—	—	Alk.	Inagglutinable by polyvalent Flexner serum; child died.
5	—	—	A. 1	A. 1 Alk.8	A. 2 Alk.7	—	—	—	Colonies very numerous; child died after passing blood and mucus for five days.
6	—	—	A. 2	—	A. 3	—	A.3	A.&Clot	Child died.
7	—	—	A. 1	—	A. 3	—	—	—	Inagglutinable by Flexner and Shiga sera; blood and mucus in stool; child recovered.
8	—	—	A. 6	A. 1	A. 1	A. 2 Alk.10	—	—	Inagglutinable by Sonne serum; child died.
9	—	—	A. 1 Alk.8	—	—	—	—	—	Inagglutinable by Shiga serum; child died.
10	—	—	A. 1 Alk.19	—	—	—	A.8	—	Inagglutinable by Shiga serum; child died.

A.3 = acid on third day; Alk. 8 = alkaline on eighth day.

Positive evidence of the pathogenicity of these bacteria cannot be adduced, but in the cases of Numbers 1, 4, 6 and 7 the occurrence of the organism in large numbers on the plates considered with the character of the stool was very suggestive. In the case of the child from whom No. 4 was isolated, the MacConkey plates yielded practically pure cultures of the organism recorded in the table and at the *post mortem* examination the appearances in the colon were those associated with dysenteric infection.

References.

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(2) F. W. Andrewes and A. C. Inman: "A Study of the Serological Races of the Flexner group of Dysentery Bacilli." Medical Research Committee: Special Report Series No. 42.

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PART II.

THE SERUM THERAPY OF INFANTILE BACILLARY DYSENTERY.

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THE next phase of the subject of bacillary dysentery in infancy and childhood to be discussed is that of the specific serotherapy of the disease and it may be stated at the outset that in the following attempt to assess the value of therapeutic antidysenteric serum the only analyses of case records included are those of children from whom the organism recovered sustained all the biochemical and agglutination reactions of dysentery bacilli of the Flexner group. It has been a matter of especial care that the obviously necessary rigid bacteriological control should be provided.

The indices of effective serum therapy might be looked for in its influence upon the fever, the duration of the disease, the number and quality of the stools and the mortality rate, the principal criterion being its capacity to effect a reduction in the death rate for any given season. That apart altogether from specific therapy the death rate occasioned by infantile bacillary dysentery varies remarkably from season to season, has already been shown (Part I.).

The age of the child is an important factor in determining death or recovery and in general children of four and five years of age and upwards do well irrespective of serum therapy.

The stage of the disease at which serum is administered, the dosage and method of administration are important considerations to each of which due weight must be attached in a computation of the results of serum treatment.

From Table IX., which embraces considerations of season and ages of the children, but takes no cognizance of the stage of the disease at which the serum was administered, it is seen that in the age group two years and under antidysenteric serum failed to effect any improvement in the therapeutic results. In the first two seasons the death rate among the serum-treated children was higher (76% and 63.6%) than that in those who were not given antidysenteric serum (54.1% and 42.8%). In the summer 1924-1925 the mortality was the same in both divisions of this age group (33.3%), but it will be noted that serum was administered to only six of the thirty children in the group.

It will be observed that in the group comprised of children over the age of two years, the four deaths which occurred were in the non-serum treated division. As serum was given to five only of the twenty-nine children in the group, it is scarcely legitimate to make a comparison of the mortality rates in the children treated by the administration of antidysenteric serum and those not so treated. It is worthy of note, however, that the mortality in this age group is represented by a figure very much lower than the best obtaining in the babies.

In the children included in Table IX. the average dose of antidysenteric serum given was one hundred and fifteen cubic centimetres. The smallest single dose employed was thirty cubic centimetres and this may be said to have been the routine amount for a single injection. A daily injection of thirty cubic centimetres until the child had received 120, 150 or

TABLE IX.

Year.	Age of Patient.	Serum Given.			No Serum Given.		
		Recovered.	Died.	Mortality.	Recovered.	Died.	Mortality.
1922-1923	Two years and under	6	19	76%	11	13	54%
	Over two years	1	0	0%	8	0	0%
1923-1924	Two years and under	4	7	63%	16	12	43%
	Over two years	4	0	0%	4	2	33%
1924-1925	Two years and under	4	2	33%	16	8	33%
	Over two years	0	0	0%	8	2	20%
1922-1925	Two years and under	14	28	66%	43	33	43%
	Over two years	5	0	0%	20	4	20%

even 180 cubic centimetres, was given in many instances.

The subcutaneous, intraperitoneal and a combination of these two routes were utilized with about equal frequency.

A careful analysis of the temperature and stool charts of the babies to whom serum was given, was undertaken in order to estimate the effect of the injection on the child's clinical condition.

In view of the fact that there was only one child among the forty-seven to whom serum was given, whose motions improved in quality and diminished in frequency immediately after the injection of antidysenteric serum, the result in this infant should perhaps be interpreted as *"post hoc."*

It also appeared that as a rule the injection of serum did not affect the temperature and in the few children whose fever temporarily subsided, the frequency and character of the stools remained unaltered.

It might have been anticipated that serum therapy would have operated to prolong life even though death was ultimately the issue. The average length of life of the children who died and who did not receive antidysenteric serum, was 8.5 days; those who, having been given serum, ultimately died, lived on an average 7.5 days. The duration of life is calculated from the day of the child's admission to hospital as representing the earliest date at which serum therapy might have been instituted.

Time of Administration.

The average day of the disease on which the first injection of serum was given is represented by the figure 6.5 and it is now to be considered to what extent the disappointing results are connected with the time of administration of the serum. Naturally every endeavour was made to institute serum therapy at the earliest possible moment and the relative lateness of the average day for the first injection, is due to the fact that so many of the infants did not come under observation until they had been ill for a week or longer.

TABLE X.

Time of Injection of Serum—Day (Inclusive).	Number of Patients.	Recovered.	Died.	Mortality Rate.
First to second	7	5	2	28.5%
Third to fifth	12	5	7	58.3%
Sixth to eighth	17	5	12	70.6%
Ninth to twelfth	10	4	6	60.0%
Thirteenth to eighteenth	1	0	1	—

In Table X. may be studied the results attending serum therapy as applied at different stages in the course of the infection.

In Table X. which includes the three seasons under discussion, the severe 1922-1923 season, the mild 1924-1925 and the intermediate 1923-1924, a striking reduction in mortality is seen in the unfortunately small group of children who received antidysenteric serum on the first or second day of the disease. The essential point to be determined, however, is whether specific therapy administered in the early stages reduced the mortality below that obtaining in the same season for children to whom serum was not administered. Table XI. is compiled with this object in view. In the last column are indicated the seasonal mortality rates among children not treated with serum.

It will be seen from both Tables X. and XI. that the lowest mortality rates appear in the group of children treated with antidysenteric serum on or before the fifth day. The severe season of 1922-1923 is the exception in this respect, but in both of the subsequent seasons the mortality among children who were given antidysenteric serum in the early stages of the disease, was reduced to a figure considerably below that obtaining in the children to whom no serum was given. The lowest mortality figure was reached in the group of six babies in the season 1924-1925 all of whom received serum on the first day of illness. Only one of these infants

TABLE XI.

Season.	Time of Injection of Serum—Day (Inclusive).	Number of Patients.	Recovered.	Died.	Mortality.	Seasonal Mortality in Non-serum Treated.
1922-1923	First to second	0	0	0	—	54.1%
	Third to fifth	5	1	4	80%	
	Sixth to eighth	10	3	7	70%	
	Ninth to twelfth	9	2	7	77.7%	
	Thirteenth to eighteenth	1	0	1	—	
1923-1924	First to second	1	—	1	—	42.8%
	Third to fifth	6	4	2	33.3%	
	Sixth to eighth	7	3	4	57.1%	
	Ninth to twelfth	1	1	—	—	
1924-1925	First to second	6	5	1	16.6%	33.3%
	Third to fifth	1	—	1	—	
	Sixth to eighth	—	—	—	—	
	Ninth to twelfth	—	—	—	—	

died. In that season serum was not given to children admitted to hospital later than the fifth day of the disease, as the experience of the two preceding seasons had shown it to be of no avail in influencing the course of the disease if its administration was delayed beyond this date.

The serum used throughout was that of the Commonwealth Serum Laboratories, issued as polyvalent against dysentery bacilli prevailing in Australia, a high grade product as judged by its agglutinating capacity, determined on numerous occasions and for successive batches of serum. In fact the therapeutic serum was employed in the agglutination tests for the identification of the Flexner dysentery bacilli preliminary to the classification of the strains by agglutination with the monovalent sera (Part I.).

The restriction of serum during the season 1924-1925 to those children who had been ill for not longer than five days, was the only particular in which selection was practised, if allowance be made for the fact that serum was not generally given to children of four and five years and upwards whose clinical condition seemed good. Otherwise serum was given to alternate children.

In epitomizing the experience attending the use of antidysenteric serum at the Children's Hospital, Melbourne, it may be stated that serum injected for the first time after the lapse of four or five days of illness was quite ineffective in reducing the mortality or modifying the course of bacillary dysentery.

Of six babies in whom serum therapy was instituted within forty-eight hours of the onset of illness, five recovered. Observations based on so small a number as six cases do not provide a sufficient basis for a definite conclusion, but from the course of the disease in the six infants referred to the suggestion may be offered that good results might be anticipated from the use of specific therapy in infantile bacillary dysentery provided the serum is given early in the course of the illness. The term "early" is to be interpreted as within the first forty-eight hours. This suggestion is supported by the efficacy of anti-dysenteric serum in the bacillary dysentery of adults and by the analogy provided by experience with therapeutic sera in other diseases. How can anti-dysenteric serum be reasonably expected to be efficacious if its administration is delayed until the child is overwhelmed by toxæmia or in the grip of a complicating bronchopneumonia?

That so few of the children here considered came under observation in the initial stages of illness is deplorable and would seem to indicate the need for propaganda relative to the recognition and serious import of bacillary dysentery in childhood. Apart from the loss of the time during which antidysenteric serum might be effective, prompt diagnosis of bacillary dysentery is essential on the same grounds as render necessary the recognition and notification of other specific infectious diseases. The practitioner, while sending a stool for bacteriological examination, should not await the report if he has reason to suspect bacillary dysentery and an opportunity to give serum under favourable circumstances. He should suspect bacillary dysentery whenever unduly frequent stools display specks and streaks of blood and may diagnose it confidently when the stool is non-fæculent and consists of blood and mucus.

Reports on the serotherapy of bacillary dysentery in infancy and childhood are scanty. In Australia F. H. Beare[1] has reported favourably on the use of antidysenteric serum in Adelaide and the experience of E. Marjory Little and Marjory J. Ross[2] in Sydney was also gratifying. In the United States of America H. C. Josephs and W. C. Daniels[3] reported that in a series of twenty children for whom a polyvalent serum was employed, no good effect was apparent.

In conclusion it is proposed to offer some suggestions for the improvement of the results attending the specific therapy of bacillary dysentery.

Prompt recognition of the disease is, of course, of fundamental importance. It may be taken that the sudden onset in an infant of a febrile illness characterized by the passage of small, frequent, non-fæculent motions consisting of mucus streaked with blood spells in 90% of cases bacillary dysentery. To a child presenting such a clinical picture serum should be given on sight and a specimen of stool forwarded for bacteriological examination without delay.

Having in mind the numerous occasions on which I have recovered *Bacillus dysenteriæ* (Flexner) from stools which in their general character were atypical and seemed to afford little prospect of such a finding, I would urge the necessity for a bacteriological examination of the stool in every case of acute diarrhœa which cannot be clearly assigned to a dietetic cause.

It is incumbent upon the bacteriologist to supply a prompt report. The means that I have adopted as a routine at the Children's Hospital, enable me to identify the dysentery bacillus in the same number of hours as are required to report on a swab from the throat with respect to the presence of the diphtheria bacillus.

The procedure is to inoculate MacConkey plates and incubate them overnight. On the following morning the plates are assembled and those showing colonies suggestive of *Bacillus dysenteriæ* selected. In the mattter of choosing likely colonies experience in the isolation of the dysentery bacillus developes a degree of confidence in the recognition of the colony which enables the worker in many instances to make the diagnosis by the macroscopic inspection of the plate. It is not suggested for a moment that this is sufficient, but inspection of the plates should afford a very good idea as to which contain dysentery bacilli and which do not.

After inspection of the plates I prepare 1:100 and 1:200 dilutions of polyvalent agglutinating

Flexner serum and deliver two or three drops into the concavity of a number of slides such as are commonly employed for hanging drop preparations. Bacteria from colonies regarded as likely to be those of dysentery bacilli are then taken on a platinum loop and stirred into the saline dilutions of serum. If the organisms are Flexner dysentery bacilli—and experience has shown that the Shiga bacillus plays no part in infantile bacillary dysentery in Melbourne (Part I.)—emulsification is followed by flocculation which is visible macroscopically and is generally indubitable after the lapse of five to ten minutes. During this interval the slide is gently oscillated. I have found that agglutination proceeds in the manner and within the time indicated with serum (Commonwealth) in dilution of 1:200 and on its occurrence a positive report with respect to *Bacillus dysenteriæ* (Flexner) is issued.

I am aware that such a manner of rapid identification is beset with traps and in the case of all the microorganisms considered in Part I. the full technique was observed for confirmation of the interim reports furnished by the abbreviated method. High titre agglutinating sera for *Bacillus dysenteriæ* are prone to affect intestinal organisms other than dysentery bacilli when employed in low dilution, but it was found that very few of the reports based on the shorter technique failed to be confirmed when the cultures were examined by sugar fermentation and extended agglutination tests.

For practical work and the gain in time with respect to serum therapy it affords, the advantages of the shorter technique more than outweigh the disadvantage involved in the risk of a small proportion of errors. By its means it is possible to report dysenteric stools as such by ten o'clock in the morning of the day following receipt of the specimen. I would emphasize that if a worker is to rely upon the method which I have outlined, he should be thoroughly familiar with the dysentery colony as it appears on the plates employed for the preliminary differentiation of the fermenters from the non-fermenters of lactose. The more experienced he is in the recognition of the colony, the smaller will be his margin of error in rapid diagnosis by the procedure outlined. He should not, of course, neglect the confirmatory examination.

Monovalent Therapeutic Sera.

In view of the fact that the investigation of one hundred and twenty-one strains of dysentery bacilli recovered from the stools of infants in three successive seasons showed a great predominance of *V* and *W* races over those denominated *Y* and *Z* as detailed in the joint communication of Miss Williams and myself, the suggestion is advanced that monovalent therapeutic sera should be prepared at least against the *V* and *W* races. The proportion of strains falling into one or other of these races was as high as 70.2% of the hundred and twenty-one strains examined.

Might not the use of a therapeutic serum prepared specifically against an individual race of dysentery bacillus be more effective than a polyvalent serum, provided that the identification of the serological race of the infecting organism did not occasion undue delay?

I suggest that it is feasible to identify the race of infecting dysentery bacillus in any given instance within twenty-four hours of the receipt of the stool.

Let the bacteriologist take his monovalent sera and subject each to absorption of agglutinin by bacterial emulsions of the other races as indicated in Part I. of this communication. He should be able to effect with one absorption a reduction of the titre of each serum for the heterologous races to a point at which it does not affect them in dilutions of 1:200. A sufficient amount of serum should be treated to insure that the absorption procedure would not have to be carried out too frequently. By this means he would provide himself with sera from which group agglutinins were for practical purposes removed, in other words, specifically monovalent sera.

A procedure such as the following might then be adopted. Organisms reacting to the interim agglutination test with polyvalent serum might be immediately sown in broth; let it be supposed that such broth cultures are made at or about ten o'clock in the morning. By three o'clock in the afternoon of the same day there should be sufficient growth for the carrying out of agglutination tests with "absorbed" sera, the dilutions of which might be prepared during the five hours of incubation of the broth cultures. As soon as growth is deemed sufficient, gas production and motility might be noted and the non-motile, non-gas producing organisms set for agglutination tests with the already prepared dilutions of "absorbed" sera. The result might be declared, possibly after one hour and almost certainly after two hours' exposure of the tubes to a temperature of 45° C. in either incubator or water bath, for by that time agglutination should have occurred in at least 1:400 dilution of the particular serum to which the strain is susceptible.

The above suggestion for the rapid determination of serological race is not put forward as infallible. Abbreviation of technique involves risk of error and the necessity for a subsequent confirmatory examination, but in the hands of a worker experienced in dysentery bacilli the scheme outlined should enable him to identify the serological race promptly and with but a small proportion of errors. If for any reason an extra day is required to establish the race, polyvalent serum might be used for the first injection.

References.

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(2) E. Marjory Little and Marjory J. Ross, "Dysenteric Conditions in Children," *Transactions of the Australasian Medical Congress (British Medical Association)*, First Session, November, 1923: Supplement to THE MEDICAL JOURNAL OF AUSTRALIA, July 5, 1924, page 472.

(3) H. W. Josephs and W. C. Davison, "The Sero-therapy of Bacillary Dysentery in Children," *The Journal of the American Medical Association*, 1921, Volume LXXVII., page 1863.

The Medical Journal of Australia

SATURDAY, DECEMBER 5, 1925.

Dichotomy.

Dr. A. Norman McArthur recently invited expressions of opinion and records of experience concerning the frequency or infrequency of fee-splitting among medical practitioners in Australia. Although a few weeks have passed since the publication of his letter, the members of the profession are silent. But even if some had responded to Dr. McArthur's invitation, very little information would have been forthcoming. Those who practise dichotomy, would not have confessed. Hearsay or indirect evidence of this ethical offence would have been valueless. Perhaps one or two might have followed Dr. McArthur's lead and have told of incidents suggesting that some practitioners are in the habit of dividing a fee with specialists. In this issue we publish a letter from a young and honourable practitioner who has obviously failed to grasp the principle on which the ethical rule has been based. As there may be many others whose attention is so rivetted to the absorbing question of endeavouring to secure adequate remuneration for services rendered, we deem it advisable to explain in some detail why dichotomy must be regarded as a dishonourable act and why those who indulge in it, should be required to give an account of their actions.

Fee-splitting means the payment by one medical practitioner of part of the fee he has received from a patient to another practitioner who has also had professional dealings with the same patient. The patient is unaware of the transaction. He has been asked to pay a fee to one practitioner and has complied with the request. Usually it is an operating surgeon who hands over to the general practitioner part of his fee. In ordinary parlance the surgeon gives to the general practitioner a secret commission. If the patient were aware of what is happening, he would naturally conclude that the surgeon has raised his fee in order that without hurting himself, he may be able to bribe the general practitioner. Moreover, since the general practitioner is also paid by the patient, the latter would regard the transaction as dishonest, for it involves double payment for one service, an open payment and a secret one. Secret commissions are always dishonourable and may be criminal. A doctor's dealings with his patient should ever be placed on the highest ethical basis. "Inquirer" may wish to regard the practice of medicine as a trade and to indulge in commercial expedients, such as the employment of fee collectors, but he can do so only by lowering the dignity of the profession and by undoing the good work which Hippocrates started and the British Medical Association has developed. He is wrong, however, in assuming that the practice of fee-splitting is held to be wrong because it tends to commercialize medicine. In ordinary commerce secret commissions are taboo.

Why is this secret commission offered to the general practitioner? That it is a bribe, intended to encourage the practitioner to recommend the surgeon to other patients, cannot be questioned. No honourable medical practitioner can justify the selection of a surgeon or consultant for his patient if this is based on a financial consideration. What would be the opinion of the patient if he knew that his trusted family doctor refers him to a surgeon for an operation, not because the particular surgeon is more experienced or more skilled in the branch of surgery involved than other available surgeons, not because his services are likely to be more valuable in the given set of circumstances than any other, but because this man has made it his habit to give a substantial bribe for the recommendation. The position is untenable. This is not the opinion of a few leaders of the medical profession whose success has rendered commercial expedients unnecessary. It is the well considered dictum of the whole medical profession in the United Kingdom and in the United States of America. The General Medical Council has condemned it. The British Medical Association regards it with abhorrence. And every medical practitioner who sees these things as they are, who wishes to raise the level of dignity and honour of his noble profession, shuns it on all occasions.

It is true that at times patients refuse to discharge their financial obligations to their medical attendants. Gratitude is rare on this earth. The upright medical practitioner should accept these disappointments in a philosophical spirit. The majority of patients pay the usual fees charged by their medical attendants. But even if a general practitioner suffers in the way mentioned by "Inquirer," he is not justified in making good his losses in an unethical manner.

Current Comment.

GASTRIC SECRETION IN NEW BORN INFANTS.

It has been known for nearly fifty years that the gastric mucosa of a four months old fœtus contains pepsin. Other digestive ferments have been found in the fœtal organism before birth. Since it is probable that digestive ferments like antibodies arise in response to antigenic stimulation, the early appearance of pepsin, rennet, lipase and amylase may be ascribed to the absorption from the maternal circulation of protein, casein, fat and starch. This antigenic action would be haphazard and to some extent irregular. Dame Nature does not as a rule waste her efforts. Since these digestive ferments are not required until after birth, it would serve no useful purpose if the function were developed early under well defined physiological principles. The glandular tissues responsible for the supply of these ferments have to be trained to a certain degree before birth so that the normal new born infant may be able to make use of the mammary secretion in the form provided in the first few days of life. That the process of training should be a prolonged and gradual one is by no means unlikely, for physiological processes are never sudden and violent like pathological processes. While the bare fact that these ferments may appear at a relatively early period has been ascertained, very little is known concerning the normal secretion of the stomach mucous membrane of new born, unfed infants. In 1872 Szydlowsky found hydrochloric acid and rennet in the stomach of an unfed infant seven hours after birth. Hess, too, found hydrochloric acid in unfed infants' stomachs. In these circumstances it is of interest to note that Dr. C. Griswold and Dr. A. T. Shohl have made some studies of the secretion of the gastric mucosa of new born infants in the New Haven Hospital.[1] They examined twenty-five infants. The gastric secretion was collected through a small, soft rubber catheter. There is reason for assuming that in every instance the whole of the contents was evacuated. The fluid was tested for its hydrogen ion concentration and for its buffer value. Normal gastric juice has an acidity of pH 1.3 to 1.8. Drs. Griswold and Shohl found that in infants between fifteen minutes and fourteen hours of age, all unfed, the hydrogen ion concentration of the gastric secretion varied between pH 1.7 and pH 4.4, with an average of pH 2.6. Five of the infants yielded a gastric secretion with pH under 2; in four the value was pH 3.0, while in one it was pH 3.2, in another pH 3.7 and in a third pH 4.4. From the fifth to the tenth days the acidity varied between pH 2.0 and pH 3.0, with an average of pH 2.5 on the fifth day and the same on the tenth day. The acidity of the gastric contents of one of the infants was determined two months after birth; it was pH 3.4.

The buffer values expressed as cubic centimetres of tenth normal sodium hydroxide per hundred cubic centimetres of secretion varied between five and fifteen cubic centimetres in unfed infants at birth or within fourteen hours, with an average of ten; on the fifth day it varied between five and thirty-five, with an average of about eleven, while on the tenth day it varied between five and twenty, with an average of approximately eleven cubic centimetres. The quantities secreted were very small. At birth they averaged 4.5 cubic centimetres with a range from two to ten cubic centimetres; at the fifth day the average was 6.5 cubic centimetres, with a range of from two to twenty cubic centimetres, while on the tenth day it averaged 4.9 cubic centimetres, with a variation from two to twenty cubic centimetres. It thus appears that in early infancy the gastric secretion is very small in amount and is considerably less acid than in after life. The acidity lessens as infancy proceeds. This finding is not in keeping with the doctrine that the major part of the digestion in early infancy is intestinal and not gastric. The secretion of acid does not seem to depend on the taking of food or other usual appetite stimulation, although Drs. Griswold and Shohl suggest that it may be reflex from sucking or from hunger. These observers have not attempted to evaluate the pepsin content of the gastric secretion. In the absence of this information it is impossible to estimate the functional capacity of the secretion at birth or to determine the mechanism of its production. With fuller data concerning the gastric contents and the records of the range of the hydrogen ion concentration of colostrum and the early milk, the digestive powers at birth could be measured with some degree of accuracy.

[1] *American Journal of Diseases of Children*, October, 1925.

SYPHILIS AS A CAUSE OF STILLBIRTH.

The frequency of syphilis as a cause of disability and death of infants has long been recognized. Syphilis is a preventible and a curable disease and yet the amount of disability and death due to it is incalculable. With one accord all authorities agree that syphilis is the commonest cause of stillbirth. Reliable statistical evidence of the number of fœtuses who succumb to this plague in their mothers' wombs is not available. But there are indications that this event is responsible for an enormous waste of valuable life. In the annual report for the year 1924 of

Sir George Newman, Chief Medical Officer of the Ministry of Health in Great Britain, it is recorded that of the 22,010 persons dealt for the first time in England and Wales during the year on account of syphilis, no less than 2,387 were suffering from congenital syphilis. This represents a frequency of 10.8%. It is unlikely that this figure is the true indication of the amount of congenital infection, for in a voluntary system, even with encouragement and stimulating propaganda, infants, children and young persons with manifestations of the disease will not seek aid at the venereal diseases treatment centres as often and as freely as persons who have acquired a venereal disease. But even if the figure be accepted as the annual incidence of congenital syphilis, the seriousness of the problem is at once apparent when it is pointed out that in addition to causing an unknown loss of life before birth, syphilis is transmitted to one person out of every 16,232 of the population each year.

The prophylaxis of congenital syphilis depends on the supervision extended to expectant mothers as well as on the attack on the disease as soon as it manifests itself. The prospects of success appear at the present time to be but moderate. It is true that the machinery provided by the *Venereal Diseases Acts* in the several States has given a stimulus to the medical profession to make a desperate effort to cope with infection wherever it exists. If all the provisions were rigidly enforced and defaulters were compelled to submit to treatment until no longer infective, better results would be obtained. Valuable aid is being rendered by voluntary societies in educating the public concerning the significance of venereal diseases. But it is a herculean task to ferret out every infected person and to enforce treatment and a still more difficult one to combat ignorance and unconcern.

We have referred on numerous occasions to the extremely instructive work of N. Hamilton Fairley and R. Fowler and have pleaded for a systematic application of the Wassermann test to the blood of all women confined in public hospitals and to the blood of their infants. If the test could be applied to the blood of all women whose pregnancies terminate prematurely after the fifth month, much information would be gathered for the purpose of preventing further intrauterine syphilis. A great deal depends on the reliability of the diagnosis of the disease in a fœtus. In order to facilitate the diagnosis, Dr. John F. Taylor and Dr. J. Forest-Smith, working on behalf of the Medical Research Council, have endeavoured to ascertain the most dependable means of distinguishing between stillbirth from syphilis and stillbirth from other causes.[1] Unfortunately they have limited their studies as far as the second class is concerned to stillbirths caused by difficult labour, *placenta prævia*, accidental hæmorrhage and other obstetric accidents. That certain infective processes other than syphilis may cause the death of the fœtus *in utero* is well known and comparative data in connexion with these less common causes would have been instructive. Be this as it may, it appears that all the stillborn syphilitic fœtuses examined by the authors were macerated at the time of expulsion. Of the sixty-six fœtuses who lost their lives from obstetric causes, thirteen were definitely macerated and five bore evidence of commencing changes. Drs. Taylor and Forest-Smith refer to the control group of fœtuses as "traumatic." While maceration may be a characteristic of syphilis in an unborn infant, it has certainly been noted in others who have died in the uterus and have not been expelled at once. Of greater diagnostic importance is the age of the fœtus at the time of birth. Forty-one of the sixty-six control fœtuses were born at full term, that is at nine months. Of the twenty-three syphilitic fœtuses only three reached the age of nine months, while nine were born at eight months, five were born at the age of seven months, four at the age of six months and two at the age of five months. While we hold that the authors should not attempt to express the frequencies in percentage figures on account of the smallness of their numbers, it is evident that a syphilitic fœtus is more likely to die between the sixth and eighth months than later. It is obvious that age of the fœtus at the time of its death and expulsion when the cause is an obstetric one, will be determined by the occasion of the accident of the pregnancy or labour. It is almost a platitude to record that the age of a fœtus lost during a difficult labour is usually nine months. In the next place they found that no indication of syphilis can be gained from the weight or length measurement of a dead fœtus or from the weight of its liver or kidneys. On the other hand they found that the spleen is usually much heavier when death has been caused by syphilis than when it was the result of an obstetric accident. Chondro-epiphysitis, as would be expected, was found to be very uncommon among stillborn infants not affected with syphilis. Only one out of the sixty-six manifested slight signs of this condition. Among the twenty-three syphilitic fœtuses eighteen were affected by this condition and in five there were slight signs. In the last place they record the results of the Wassermann test applied to the blood serum of the mothers. The test was applied to seventeen mothers who had given birth to dead syphilitic infants. In twelve a reaction was obtained. The serum of five failed to react. A second test was carried out with the serum of two of these mothers at a later date, but no reaction was obtained. None of twenty months who lost their babies as a result of an obstetric accident, yielded a reaction to the test. It appears that the fœtuses of five of these women were macerated. As a result of these observations Drs. Taylor and Forest-Smith conclude that when the weight of the spleen represents more than 0.45% of the total body weight, when chondro-epiphysitis is present and when the mother's serum reacts to the Wassermann test, the cause of the stillbirth is probably syphilis. If the fœtus is not macerated and if it reaches full time, it is probable that its death has not been due to syphilis.

[1] *The Journal of Obstetrics and Gynæcology of the British Empire*, Autumn Number, 1925.

Abstracts from Current Medical Literature.

PHYSIOLOGY.

Asynchronism in the Right and Left Ventricles.

It is generally assumed that the two ventricles initiate and terminate their contractions simultaneously. This question was investigated by L. N. Katz (*American Journal of Physiology*, May, 1925). The two intraventricular pressures or the two aortic pressures just outside the semilunar valves were recorded simultaneously by optical manometers. It was found that during normal experimental conditions right and left ventricular systoles and also their ejection phases neither begin nor end simultaneously and as a rule are of unequal duration. Sometimes the right, sometimes the left ventricular systole or ejection preceded the other. In only two cases out of twenty-four experiments did contractions begin simultaneously in the two ventricles. The duration of the contraction process varied independently in the two ventricles. The period of ejection was, however, longer on the right than on the left side. The phasic relations of the contractions in the two ventricles are readily altered by vagal influences. Infusion of saline solution caused a lengthening of both ventricular systoles, but sometimes the effects were greater on the right, at other times on the left side. It follows that changes in one ventricle do not necessarily indicate the changes in the other. The *T* wave in the electrocardiogram is explained by the asynchronous cessation of right and left ventricular contraction.

Effect of Pituitary Extract on Secretion of Urine.

The data so far available as to the action of pituitary extract on the secretion of urine appear on the surface to be of a conflicting nature. With anæsthetized animals some observers have noted a diuresis, others a diminished rate of secretion. R. L. Stehle and W. Bourne (*Journal of Physiology*, July, 1925) have attempted to obtain additional information as to the action of pituitrin. They employed for the most part normal, fasting unanæsthetized dogs with bladder fistulæ and compared the urine in regard to quantity and composition before and after intravenous injection of pituitary extract. If the urine secretion is proceeding slowly, the administration of pituitary extract causes first complete anuria for varying length of time—a few minutes up to fifteen or twenty minutes—and then a period of diuresis followed by a return to normal. A volume increase is nearly always present. Along with increase in volume rate there is an extremely striking effect on the chloride excretion; this has amounted to as much as a seventy-four-fold increase in the minute rate as compared with the chloride control rate. Urea and phosphate excretion are also increased in unmistakable fashion, but the quantitative effect is much less than in the case of chlorides. The action on the volume rate may be merely a secondary one, the primary effect of pituitary extract being on the chloride secretion. The first portion of urine passed after the period of anuria contains protein. In the anæsthetized animal there is a slight diuresis, but the chloride excretion is very slightly affected. If the urine secretion is proceeding rapidly, the increase in the volume rate is absent. The volume rate after pituitary extract never reached the control rate. The chloride excretion was, however, greatly increased. It may be assumed that the pituitary gland has a definite function in determining the secretion or conservation of water and chlorides and it may well be that this control is exerted over other constituents of the urine as well. The locus of action is probably in the kidney itself. Changes in the water and chloride content of the blood do not appear to be involved.

The Action of "Insulin" on Erythrocytes.

The means whereby "Insulin" lowers the level of the sugar in the blood has been the subject of a large amount of work, but is by no means understood. Haldane, Kay and Smith as a result of an investigation into the effect of "Insulin" on the blood volume suggest that definite changes in the permeability of certain of the body cells may be brought about by the action of "Insulin." J. Secker (*Journal of Physiology*, September, 1925) has studied the effect of "Insulin" on the permeability of the red corpuscle to glucose and chloride. The influence of the salts of guanidine on permeability was considered in conjunction with the influence of "Insulin," because of the suggestion of a close relation between these two substances. He estimated the glucose and chloride content of whole blood and of plasma or serum from the blood. To another sample of the same blood was added some "Insulin" and after an interval the plasma or serum separated and the glucose and chloride contents were determined. Glucose or chloride was also added to blood together with "Insulin." The addition of "Insulin" decreased the amount of glucose and chloride in the plasma or serum without altering the quantities in the whole blood, indicating a passage of these substances into the corpuscles. It is not unjustifiable to assume that "Insulin" has a similar action on the permeability of the tissue cells; the fall of blood sugar without a concomitant increase in sugar oxidation observable after the injection of "Insulin" may be due to the passage of sugar into the tissues, especially muscle. Guanidine has a similar action to "Insulin" provided that calcium be present. Since excision of the parathyreoids causes an increase of guanidine and a decrease of the calcium present in the blood, the facts suggest a method by which the parathyreoids may influence carbohydrate metabolism. Further, if, as Burns finds, "Insulin" causes a liberation of guanidine, the guanidine with the existing calcium will reinforce the "Insulin" hypoglycæmia.

Physiology at Great Heights.

T. H. Somervell (*Journal of Physiology*, September, 1925) gives some data on observations made on the members of the 1924 Everest expedition. Up to 16,500 feet there was not much change in the percentage of carbon dioxide in the alveolar air nor in the alveolar respiratory quotient. At 23,000 feet the alveolar air of four subjects showed an average carbon dioxide percentage of only 2.78 instead of about 6. The alveolar respiratory quotient averaged only 0.38. At a great height breathing is so rapid (about fifty respirations to the minute) that the carbon dioxide is washed quickly out of the alveoli and hence in this series the percentage of carbon dioxide is naturally very small. The pulse during actual motion upwards was found to be beating 160 to 180 per minute, sometimes even more; it was regular in rhythm and of good volume. All who had gone over 27,000 feet were found to have dilated hearts which took one to three weeks to recover. The respiration was about 50 to 55 per minute while climbing. Approaching 28,000 feet for every single step forwards and upwards seven to ten complete respirations were required. Breathing quickly and deeply is very easy at a great height owing to the low density of the air. At the base camp at 16,500 feet the hæmoglobin values for the members of the party varied from 114% to 142%. Three weeks at 21,000 feet had no apparent effect on the hæmoglobin value. The hæmoglobin values of two Tibetans, taken at 16,500 feet, at which height most of their lives had been spent, were 92 and 82, remarkably low figures for men who could race up steep slopes about twice as fast as the white members of the party.

Deprancreatized Dogs and "Insulin."

I. L. Chaikoff, J. J. R. Macleod, J. Markowitz and W. W. Simpson (*American Journal of Physiology*, September, 1925) have studied the effects of cessation of "Insulin" treatment in thin and fat depancreatized dogs kept alive by "Insulin" and the effects of subsequent treatment with "Insulin." They found that the blood sugar of thin diabetic dogs was decidedly lower than in fat dogs. The ketone bodies were also higher in fat as compared with thin dogs. In fat dogs acetoacetic acid was relatively more plentiful than β-hydroxybutyric acid and in thin dogs oxybutyric acid was relatively more plentiful than diacetic. Few depancreatized animals that have been kept alive by means of "Insulin," can survive its withdrawal for more than five days, the risk in fat ones

being much more serious in this regard than in emaciated ones. The fatal symptoms in fat animals are very like those of severe coma in man. The fact that critical symptoms supervene in fat diabetic dogs more quickly than in thin ones on withdrawal of "Insulin" corroborates the clinical experience that diabetic patients who have put on much fat under "Insulin," are liable to develope alarming acidosis when its use is discontinued. When "Insulin" is given, there is a striking parallelism in the fall of blood sugar, inorganic phosphate and β-hydroxybutyric acid which suggests that they may go to form a common product. The D: N ratio in the urine declined steadily in thin animals following the withdrawal of "Insulin" and food, to far below the 2.8 level generally considered indicative of derivation of glucose from protein.

BIOLOGICAL CHEMISTRY.

Relations of Hæmoglobin.

R. HILL (*Biochemical Journal*, December, 1924) has prepared a series of metallic derivatives of hæmoglobin and has studied their absorption spectra. From a consideration of the spectra of these pigments and of the effect of treatment with alkali of hæmoglobin the author advances certain hypotheses in respect to the structure of these pigments. The metallic derivatives of hæmatoporphyrin with different metals present absorption spectra of three types in which the absorption bands resemble those of acid hæmatoporphyrin, oxyhæmoglobin and hæmachromagen. These types of absorption bands are independent of the valency of the metal in combination as can be seen by noting the difference between the derivatives with silver and with sodium and the similarity of the derivatives with zinc, aluminium and tin. The author suggests that a simple explanation can be found by supposing the spectrum to be an indication of the coordination number of the metal with reference to the pigment. Thus in hæmoglobin iron has a double linkage with the pigment, in oxyhæmoglobin four linkages with pigment and in hæmochromagen six linkages with pigment. Photographs of the spectra of different metallic derivatives show the evidence for these suppositions.

Gastric Urease.

J. M. LUCK AND T. N. SETH (*Biochemical Journal*, March, 1925) have made further investigations into the physiology of the urease found by them in quantity in the mucous membrane of the stomachs of *carnivora*. They have completed a series of experiments on living animals designed to show that the enzyme behaves similarly *in vivo* to *in vitro*. An increased concentration of urea in the cells of the gastric mucous membrane has been invariably accompanied by an increase in the amount of ammonia in the venous blood from the stomach. The authors have made estimation of ammonia in samples of blood from the portal vein and from the arterial system before and after placing urea, hydrochloric acid and ammonium chloride in the stomach. Under these conditions it was found that urea and ammonium chloride lead to a definite increase of the ammonia in the blood from the portal vein. Under no circumstances has any increase in the amount of urea in the blood in the portal vein been noted.

Oxidation of Fatty Acids.

P. W. CLUTTERBUCK AND H. S. RAPER (*Biochemical Journal*, March, 1925) have made a study of the oxidation of the ammonium salts of stearic acid, palmitic acid, myristic acid and other normal fatty acids with the object of obtaining an insight into the mode of oxidation of fats within the animal body. It is well known that Dakin obtained β-keto acids and ketones by the oxidation of ammonium salts of normal saturated fatty acids with hydrogen peroxide. This observation attracted notice since it gave support to the theory of β-oxidation of fatty acids in the animal body. Later Dakin showed that phenyl propionic acid in the animal body gave rise to β-keto acid, β-hydroxy acid and unsaturated acids. The authors have tried to obtain unsaturated acids by oxidation *in vitro*. The results of this investigation show that the oxidation with hydrogen peroxide is not confined to the β carbon atom, but also takes place at the γ and δ carbon atoms. The authors have isolated compounds which prove undoubtedly that γ and δ-keto acids are formed in the oxidation. They have also observed that no hydroxy acids are formed in the oxidation. They suggest that hydroxy acids obtained *in vivo* are derived secondarily from keto acids rather than by direct oxidation. They point out the necessity of renewed investigation into the occurrence of γ and δ oxidation in the animal body.

Avian Deficiency Disease.

T. OGATA, S. KAUAKITA, S. SUZUKI AND S. KAGOSHIMA (*Mitteilungen aus der Medizinischen Fakultät der Kaiserlichen Universität zu Tokyo, Band* XXXII., *Heft* 3, March 31, 1925) state that the disease condition produced in birds by a diet of polished rice is due to deficiency of vitamin *B*. It can be produced by diets that are sufficient in all respects excepting for vitamin *B* and it begins to appear when the reserve of vitamin *B* in the body has become exhausted. This diseased condition is closely connected with carbohydrate metabolism. If the carbohydrate of the food is diminished and the protein (casein) increased, the onset of the symptoms is delayed. When the deficiency of vitamin *B* is only partial, the ultimate result is the same as in absolute deficiency, but the incubation period is longer. The disease syndrome presents two stages. In the first the characteristic sign is the development of hyperglycæmia. Death may occur in this stage. The second stage is marked by the development of paralysis. During the stage of incubation there is a gain in weight, but in the more extreme cases of vitamin deficiency this gain is short and is soon followed by a loss. In the more prolonged incubation periods exhibited by birds suffering from lesser degrees of deficiency, there is a definite and progressive gain in weight from increase of fat. This ceases when the stage of hyperglycæmia begins and when the stage of neuritis arrives there is a rapid loss of weight. There are no changes in the nerves observable in birds which die during the stage of hyperglycæmia. Coincidentally with the appearance of hyperglycæmia there developes a very profound degree of anæmia together with a lymphopenia and corresponding changes in hæmopoietic organs. The authors point out certain features which in their opinion contradict the current belief that human beri beri and the avian disease are identical in their nature and causation, though they admit that the dietary of beri beri patients is deficient in vitamin *B*. As examples of difference, they point to the frequent occurrence of convulsions and to the prominent ataxic symptoms that occur in the avian disease, but not in human beri beri. They contend further that the grave circulatory disturbances that occur in beri beri, are not seen in birds and form another conspicuous distinction. Again, when œdema is seen in birds, it seems to be secondary to anæmia and to renal changes, but the œdema occurs in nearly all cases of beri beri and the authors consider it to be due to peripheral causes.

Hæmoglobin and Iron in Blood.

THE estimation of hæmoglobin is a very unsatisfactory procedure because of the lack of a uniform standard in common for all methods of determining hæmoglobin. The majority of hæmoglobinometers are graduated to read in percentages, 100% being used as the normal standard; but in each apparatus a different number of grammes corresponds to 100%. G. E. Sackett (*Journal of Laboratory and Clinical Medicine*, September, 1925) has determined the iron content and from it the number of grammes of hæmoglobin per hundred cubic centimetres of blood of fifteen apparently normal men and fourteen apparently normal women and also the number of red corpuscles per cubic millimetre of the blood of the same individuals. Haldane has taken as 100% blood with an oxygen capacity of 18.5 cubic centimetres corresponding to 13.8 grammes of hæmoglobin per hundred cubic centimetres. Newcomer has recently taken 16.92 grammes of hæmoglobin per hundred cubic centimetres of whole blood as his standard of 100. From the iron content of the blood Sackett found that 16.4 grammes of hæmoglobin corresponds to a five million red cell count and he takes this as the normal standard.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, Adelaide Street, Brisbane, on September 4, 1925, DR. VAL. MCDOWALL, the President, in the chair.

Popular Medical Fallacies.

DR. ANDREW STEWART read a paper entitled: "Popular Medical Fallacies" (see page 645).

DR. A. JEFFERIS TURNER referred to the folly of sending patients with pulmonary tuberculosis out west, particularly if they were young people. Patients suffering from the disease in a chronic form might do well if they were in good financial circumstances. Most of the patients were not under control and sanatorium treatment would be much better for them. The climate of Brisbane in winter was very good for tuberculosis, but the same could not be said of it in the summer. It was best to get the patient in the early stages and to put him under the most favourable conditions. He considered that treatment by the production of an artificial pneumothorax gave very good results in suitable cases, but that it should not be employed for patients who were suffering from the disease in an advanced stage.

DR. A. H. MARKS, O.B.E., D.S.O., stated that when examining Imperial pensioners he had been struck by the fact that many of them had been told to come out to a warm dry climate, such as that of Australia. He suggested that Dr. Stewart was a suitable person to write officially to *The British Medical Journal* and *The Lancet*, warning practitioners in England of the conditions which obtained in Brisbane.

DR. D. GIFFORD CROLL, C.B.E., said that he thought that X ray examination of the chest was frequently very useful. If a careful physical examination of the chest was made first and if the results obtained were compared with the film, the suspected areas would often be found to coincide. He considered hæmorrhage to be an accident in the course of the disease. It did not necessarily show an exacerbation of the trouble. He always gave "Hæmostatic Serum" at once to check the immediate hæmorrhage and he kept the patient quiet for a week or two to obviate its recurrence. The loss of blood frequently caused a rapid spread of a latent focus. The problem of obtaining suitable work for patients suffering from arrested pulmonary tuberculosis had been made much more difficult by industrial conditions following on the adoption of a basic wage.

DR. L. M. MCKILLOP could not understand the physiology of the statement that vesicular murmurs were due to the larynx. He had heard murmurs in the opposite lung when one had been affected as well as the larynx.

DR. J. LOCKHART GIBSON considered that there must be a fallacy in the experiment quoted by Dr. Stewart regarding the causation of vesicular murmurs. In the case quoted there could be no resistance to the output of air and therefore there could be no rustling of air in the vesicles.

DR. VAL. MCDOWALL after thanking Dr. Stewart for his paper, said that he thought Dr. Stewart had condemned a warm, moist climate. He asked Dr. Stewart how he could explain the fact that life on the sea coast undoubtedly benefited some patients. If the patient was in good circumstances, the warm, dry interior of Queensland was most suitable. He thought that Dr. Stewart was too hard on the radiologists. Cavities of smaller size could be picked out more easily by radiological than by physical examination. He emphasized the need of considering the history of a patient before expressing an opinion on the radiological appearance of his chest. The most unsatisfactory film would give more information than the use of the screen alone. He always used both methods in examining chest conditions. The screen examination alone did not reveal any of the fine details.

A MEETING OF THE EYE AND EAR SECTION OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Eye and Ear Hospital, Melbourne, on August 25, 1925, DR. FRANK ANDREW, the President, in the chair.

Clinical Evening During the Post-Graduate Course.

It was announced that the Eye and Ear Section proposed to organize a clinical evening on November 19, 1925, during the holding of the post-graduate course. Members of the Branch would be invited to attend.

Deep Infection Leading to Loss of Eyes.

DR. LEONARD MITCHELL read a paper on the loss of eyes as a result of deep infection during systemic diseases. He stated that three patients at the Melbourne Hospital had lost an eye from some constitutional disease since the beginning of the current year. The first was a girl of eighteen years who had been admitted with headache and pain in the left side of the chest. The fundi had been found to be healthy. Lumbar puncture had yielded turbid fluid under increased pressure. The fluid had contained pus cells in great numbers and meningococci. Three days after admission the left eye had become inflamed. At first it had been thought that the condition was due to ether which had been used for the daily anæsthetic for the lumbar punctures. Two days later the eye had been examined by a member of the ophthalmological staff who had reported that a purulent conjunctivitis and iridocyclitis was present, probably a panophthalmitis due to a central rather than to a peripheral infection. The cornea had been found to be uninvolved. The eye had become worse and had ruptured spontaneously on the eighth day. It had been arranged that the eye was to have been excised on that day. The ruptured globe had been excised by Dr. Ethel Parnell. The socket had healed well. The patient was still in hospital.

The second patient was a single woman, aged twenty-six years. She had been delivered seven weeks before admission and a pyelitis had followed the parturition. Cough and the expectoration of rusty sputum had ushered in a pneumonia which had not resolved. The bacteria isolated were *Staphylococcus aureus* and a Gram-positive bacillus of the coliform type. Resection of a rib had been performed six weeks after admission. Hypopion had developed and other signs of cyclitis had been noted four weeks after admission. The eye condition had not responded to atropine and foments. Panophthalmitis had supervened. The eye had been enucleated by Dr. Mitchell seven weeks after admission. After a considerable time the general condition had improved and the patient had been discharged four and a half months after admission.

The third patient was a married woman, aged thirty-three years. While this patient was attending the gynæcological clinic for gonorrhœa, she had visited the eye department because her right eye had been swollen and inflamed for three days. The eye was almost sightless. The pupil was blocked, there was a small hypopion and much chemosis of the lids. The tension of the eye was much increased and the globe was of stony hardness. She had been admitted to hospital with a condition of absolute glaucoma. A scleral puncture had been performed under anæsthesia as a preliminary measure. It had been Dr. Mitchell's intention to trephine the eye later, but the puncture had led to the escape of a free flow of pus. The eye had then been removed by Dr. Frank Miller. After removal a section of the eye had been made and it had been found that there was an enormous mass of white plastic material covering the whole of the ciliary body. This material had proved sterile on culture; this lent strong support to the diagnosis of a gonorrhœal infection of the eye.

Chronic Paranasal Sinusitis.

DR. E. GUTTERIDGE read a paper on a series of cases of chronic paranasal sinusitis treated with colloidal manganese. He pointed out that he had undertaken the investigation to determine the effect of colloidal manganese in the treatment of this condition. Watson Williams had reported that he had employed colloidal manganese in antral and other suppuration by injection into the subcutaneous

tissue, intermuscular septa and periosseous tissue. Martindale had announced in the "Extra Pharmacopœia" that colloidal manganese had a special affinity for the staphylococcal group. Dr. Sydney Pern had claimed good results in chronic sinusitis in THE MEDICAL JOURNAL OF AUSTRALIA (May 9, 1925, page 481), but Dr. W. Kent Hughes had challenged its efficacy in a later issue (June 6, 1925, page 611).

Dr. Gutteridge stated that there were possible uses for such a drug: (i.) In mild cases in which neither the signs nor the symptoms warranted operative interference, provided that there was good drainage through the natural channels; (ii.) in more severe cases as the sole form of treatment; (iii.) in cases in which appropriate operative treatment had been employed, some of the sinuses having been opened or treated radically. In his series nineteen patients had been treated. Two had failed to return for control. Some of the patients had complained of pain, local discomfort and swelling of the arm. The local reaction at times had been very severe with redness and œdema. The majority of patients had complained of malaise, headache and some of them of nausea and vomiting. Three patients had definite signs of paranasal sinusitis, consisting of inflammation and œdema of the middle conchæ and of the posterior end of the inferior concha and swelling of the posterior part of the septum. The diagnosis had been confirmed by skiagraphic examination. In some patients the antrum had been washed out and pus had been removed. The usual preliminary routine treatment with menthol ointments and collunaria had been given.

Dr. Gutteridge divided his series into two groups. In the first the patients had definite chronic paranasal sinusitis and all had been treated previously by operation. The injections had been given into the deltoid muscles. There were eight patients in the group. In four of the patients no improvement followed; in two there was a gradual improvement which Dr. Gutteridge ascribed to the operative treatment and in the remaining two it was doubtful whether there was any improvement. At the end of three months there was no undoubted improvement in all of the patients.

The second group comprised nine patients. Five patients had had no operative treatment on account of the sinusitis, but two had had a submucous resection. Four patients had had operative treatment. These patients had received one cubic centimetre, followed by weekly injections of two cubic centimetres for three weeks and thereafter one cubic centimetre each week.

Dr. Gutteridge stated that the results were disappointing. Among seventeen patients treated for all varieties of chronic paranasal sinusitis only one manifested any improvement. Even in this patient there was still pus in the antrum.

In the discussion that followed, several members expressed their agreement with Dr. Gutteridge's conclusions. All the members seemed to be sceptical concerning the statements that had been made in favour of treatment with colloidal manganese for sinusitis.

Tuberculosis of the Larynx and Pharynx.

DR. FRANK ANDREW exhibited a specimen of a larynx on which Irwin Moore's operation had been performed. The patient had died as the result of an accident during a subsequent operation on the thyreoid gland.

Atrophic Rhinitis.

DR. C. M. EADIE presented a patient with atrophic rhinitis which had been treated by the insertion of a small implant of rib on each side of the septum. Considerable improvement had resulted.

Pharyngeal Neoplasm.

DR. H. FLECKER showed a patient whom he had treated by means of a single dose of deep X ray therapy for a neoplasm of the pharynx. The treatment had been successful.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been elected a member of the Victorian Branch of the British Medical Association:

Hartnett, Francis Timothy, M.B., B.S., 1925 (Univ. Melbourne), 130, Westbourne Grove, Northcote.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

AT a recent meeting of the Senate of the University of Sydney a letter was received from Professor J. T. Wilson (Cambridge) covering the report of the Committee appointed by the Senate to consider the selection of a suitable occupant for the Chair of Anthropology in the University of Sydney.

The Committee of Selection consisted of Professor J. T. Wilson, Professor of Anatomy in the University of Cambridge; Professor Grafton Elliot Smith, Professor of Anatomy in the University College, London; Dr. A. C. Haddon, Reader in Ethnology, University of Cambridge.

After carefully considering all the possible candidates likely to be available for this important position, the Selection Committee recommended that the chair be offered to Professor A. Radcliffe-Brown, M.A., at present Professor of Social Anthropolgy in the University of Cape Town, South Africa. This recommendation was unanimously adopted by the Senate and the offer in accordance with the recommendation was made by cable. Professor Radcliffe-Brown has now signified his acceptance of the offer. The first occupant of this chair has been engaged in anthropological field work for the past sixteen years in the Andaman Islands, Australia and South Africa. The establishment of this chair has been made possible by the action of the Commonwealth and the several State Governments on the strong recommendations of the Australian National Research Council, the Universities and scientific bodies in the various States in providing sufficient funds for the upkeep of the Department.

Mr. A. Radcliffe-Brown was a scholar of Trinity College, Cambridge, and after taking his degree was awarded the Anthony Wilkin Studentship to make investigations on the Andaman islanders. As a result of this research he was elected a Fellow of Trinity College. He published an essay on the religion of the Andaman islanders in "Folk-Lore," 1909. Subsequently he was reappointed to a studentship to enable him to begin field work in north-western Australia in 1910, some of the important results of which are recorded in *Man*, 1910, 1912, 1914; *Customs of the World*, 1912, Chapter IV. dealing with Australia; *Journal of the Royal Anthropological Institute*, 1913, 1918 and 1923; *Anthropos*, 1914. In addition Mr. Radcliffe-Brown has published a monograph on "The Andaman Islanders" (Cambridge University Press, 1922) and in his presidential address to the Section of Anthropology of the South African Association for the Advancement of Science (*South African Journal of Science*, 1923) he has defined his attitude to the general problems of anthropology.

Before leaving Australasia, Mr. Radcliffe-Brown did some field work in Queensland and Tonga.

During the four years of his professorship in Cape Town he has built up a vigorous and successful department of anthropology and has stimulated widespread interest in anthropology among university students, the number of undergraduates taking anthropolgy having grown from sixteen in 1922 to forty-four in 1924; in the latter year graduate students were working at anthropology for Master's and Doctor's degrees. In addition Professor Radcliffe-Brown has given courses of lectures on native law for law students, special courses on the native problem from the economic point of view for students of the Faculty of Commerce, special courses for magistrates and native chiefs of the Transkei Territories and several vacation courses for Government officials and missionaries. Professor Radcliffe-Brown has proved himself to be a most efficient and stimulating teacher and beyond the walls of

his University has performed services to the community which have been keenly appreciated by the administrators of the Transkei Territories.

In the words of Professor Wilson: "The University of Sydney is fortunate in securing as the first occupant of the new chair one so eminently qualified for anthropological work in Australia as Mr. Radcliffe-Brown appears to be."

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION), DUNEDIN, 1927.

THE Honorary General Secretary of the Second Session of the Australasian Medical Congress (British Medical Association), Dunedin, 1927, has forwarded to us the following lists of Honorary Local Secretaries and Presidents of Sections. He calls attention to the fact that the offices of Presidents of Sections have now been filled.

Honorary Local Secretaries.

New South Wales.—Dr. F. Brown Craig, 149, Macquarie Street, Sydney.
Queensland.—Dr. R. Marshall Allan, M.C., B.M.A. Building, Adelaide Street, Brisbane.
South Australia.—Dr. R. L. Thorold Grant, King's Mead, Brougham Place, North Adelaide.
Tasmania.—Dr. E. Brettingham Moore, 149, Macquarie Street, Hobart.
Victoria.—Dr. F. L. Davies, Medical Society Hall, East Melbourne.
Western Australia.—Dr. A. S. Johnson, 252, St. George's Terrace, Perth.

We understand that Dr. R. Marshall Allan has recently resigned his position as Honorary Local Secretary on account of his appointment as Director of Obstetrical Research in Melbourne. As soon as a successor has been appointed, an announcement will be made in these columns.

Presidents of Sections.

Section I.—*Medicine:* Professor A. E. Mills (New South Wales).
Section II.—*Surgery:* Dr. B. Kilvington (Victoria).
Section III.—*Obstetrics and Gynæcology:* Dr. J. W. Dunbar Hooper (Victoria).
Section IV.—*Pathology and Bacteriology:* Dr. A. H. Tebbutt (New South Wales).
Section V.—*Preventive Medicine:* Dr. Harvey Sutton, O.B.E. (New South Wales).
Section VI.—*Ophthalmology:* Dr. A. M. Morgan (South Australia).
Section VII.—*Otology, Rhinology and Laryngology:* Dr. R. H. Pulleine (South Australia).
Section VIII.—*Neurology:* Dr. Oliver Latham (New South Wales).
Section IX.—*Diseases of Children:* Dr. A. Jefferis Turner (Queensland).
Section X.—*Naval and Military Medicine and Surgery:* Dr. G. W. Barker, C.B., C.M.G., D.S.O. (Western Australia).
Section XI.—*Orthopædics:* Dr. N. D. Royle (New South Wales).
Section XII.—*Radiology:* Dr. S. S. Argyle (Victoria).

INTERNATIONAL CONGRESS OF SURGERY.

THE next International Congress of Surgery will be held in Rome during the month of April, 1926. We are advised by Thomas Cook and Son that the steamers leaving Australia in February, March and April are filling very rapidly. They therefore suggest that practitioners purporting to attend this Congress should communicate with them in order that arrangements for their passages may be made.

THE BRITISH PHARMACOPŒIA.

THE Secretary of State for the Colonies has advised the Chief Secretary and Minister for Health in the State of Victoria that the "British Pharmacopœia" is about to be revised.

By courtesy of the Chief Secretary the Council of the Victorian Branch of the British Medical Association has been invited to make suggestions. The Council suggests that any member of the Branch who has any suggestions to offer, should forward them to the Convenor of the Scientific Subcommittee of the Branch.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned have been registered under the provisions of the *Medical Act*, 1912 and 1915, as duly qualified medical practitioners:

Dalgarno, Marjorie Clare, M.B., Ch.M., 1925 (Univ. Sydney), Upper Cliff Road, Northwood, Lane Cove.
Egan, John, M.B., B.Ch., 1923 (National University of Ireland), 52, Doncaster Avenue, Kensington.
Muller, Raymond Albert, M.B., Ch.M., 1925 (Univ. Sydney), 263, Elizabeth Street, Sydney.

Change of Name.

Sams, Clara Murray, to Wilson, Clara Murray.

VICTORIA.

THE undermentioned have been registered under the provisions of the *Medical Act* 1915, as duly qualified medical practitioners:

Guilfoyle, Denis Paul, M.R.C.S., 1915 (England), L.R.C.P. (London), Victoria Coffee Palace, Collins Street, Melbourne.
Guymer, Arthur Howes, M.B., B.S., 1915 (Univ. Adelaide), F.R.C.S., 1921 (Edinburgh), 4, Raglan Street, Ballarat.
Jamieson, George Arthur, M.B., B.S., 1925 (Univ. Melbourne), 15, Carnarvon Road, Caulfield.
McInnes, Ian Donald, M.B., B.S., 1925 (Univ. Melbourne), corner of Hutton Street and Saint George's Road, Thornbury.

QUEENSLAND.

THE undermentioned have been registered under the provisions of the *Medical Act* of 1867, as duly qualified medical practitioners:

Bendeich, Joseph Henry, M.B., Ch.M., 1921 (Univ. Sydney), Bazaar Street, Maryborough.
Broben, James Alfred, M.B., B.S., 1924 (Univ. Melbourne), Institute of Tropical Medicine, Townsville.
Douglas, Ralph Edmonds, M.B., 1925 (Univ. Sydney), Sherwood Road, Toowong.
Geaney, Milton, M.B., Ch.M., 1921 (Univ. Sydney), M.R.C.S. (England), L.R.C.P., 1924 (London), F.R.C.S., 1925 (England), South Brisbane.
Gibson, Walter Lockhart, M.B., Ch.M., 1924 (Univ. Sydney), Ipswich Hospital, Queensland.
Parry, Trevor Alexander, M.B., B.S., 1925 (Univ. Melbourne), Fitzroy Street, Rockhampton.
Reisz, Lawrence Ruth, M.B., Ch.M., 1925 (Univ. Sydney), Deighton Road, South Brisbane.
Trainor, Desmond Coleman, M.B., Ch.M., 1925 (Univ. Sydney), Saint Vincent's Hospital, Toowoomba.

Wait, Leslie Proud, M.B., B.S., 1925 (Univ. Melbourne), Ipswich Hospital, Ipswich.
Blackburn, John Herbert, L.R.C.P. & S, (Edinburgh), L.F.P.S., 1918 (Glasgow), Mount Mulligan.
McKeon, Michael Leonard Devaney, M.B., Ch.M., 1925 (Univ. Sydney), Barolin Street, Bundaberg.

Correspondence.

FEE-SPLITTING.

SIR: As Dr. Norman McArthur suggests correspondence on the subject of fee-splitting in Australia, I should be grateful if you would find space for my contribution.

As a young and therefore somewhat ignorant practitioner, I desire information as to why there is such a great objection to fee-splitting. Though I have a natural abhorrence of all wrong acts, yet fee-splitting does not suggest itself to me, so far, as in the least wrong. Not that the opportunity has ever come my way. Being a young suburban practitioner, those who can afford to pay for operations go to city surgeons directly, and those of my patients who have needed operations, go to the free public hospitals. I do not want an explanation that fee-splitting "commercializes medicine." If we were compensated for our services by the State, then we could carry on our work without a thought of payment; but so long as we need to make a living by payment received from patients directly and employ account forms and debt collectors, then the practice of medicine must be considered not only an art and science, but also a business—and is so considered by the public. "To teach the patient his financial duty to the physician" sounds nice, but how is one to do it in practice? I consider it *infra dig.* to refer to the fee during a consultation and often accounts sent to patients are ignored.

I know an elderly practitioner in a country township who attended a child during an illness of three months' duration. After two months of such attention the child's parents considered it advisable to bring over a children's specialist from Melbourne. He confirmed the local doctor's treatment and left with a cheque for fifty guineas (consultation and trip). The local doctor sent in a bill for thirty guineas for three months' daily attendance, at which there was an outcry, and after much wrangling, was eventually paid twenty guineas. I, myself, have sent in an account for two guineas for six visits and received a protest from the patient who, nevertheless, paid this sum for one visit to a city physician.

A comparison of the incomes of general practitioners who do no major surgery (whether country or suburban) with this of surgeons and consultants, show that the latter could afford to split their fees by half and still enjoy double the income of the general practitioner. Besides, such surgeons who do split their fees, enjoy a fine income. So that fee-splitting seems to me only a case of "live and let live."

I would like to suggest to our professional leaders to spend their surplus energies in (1) taking active steps to suppress unqualified practice, herbalists and other quacks; (2) considering in what way they can give a helping hand to the struggling "G.P."

Yours, etc.,

"INQUIRER."

Melbourne,
November 10, 1925.

Obituary.

JAMES McLEOD.

THE death of James McLeod, of Hurstville, which we announced in our issue of November 14, 1925, has created a feeling of profound regret throughout the entire district. Although it was widely known that his health had been unsatisfactory for some time, the majority of his friends were quite unprepared to receive the news of his sudden death.

James McLeod was born at Tain in Ross and Cromarty, Scotland, in the year 1861; he was consequently sixty-four years of age. He was educated at the Royal Academy of the ancient borough of Tain and in the course of time entered the University of Edinburgh as a medical student. He took his degrees in medicine and surgery in the year 1877. After graduation he worked as assistant to a Dr. Pitt in Chelmsford, England, for about eighteen months. In January, 1890, he determined to seek fresh fields and embarked for Australia. His registration in New South Wales is dated May 14, 1890. At first he went to West Wallsend, but he did not remain for many months. He then settled in Hurstville, near Sydney, where he conducted a very large practice. In recent years this practice has been shared by two other practitioners, Dr. A. C. Thomas and Dr. C. G. McDonald. His ripe experience based on a sound knowledge of his profession rendered him a very valuable general practitioner. He was a very modest and retiring man and was generous to a fault. He commanded the confidence as well as the affection of his patients and the admiration of his colleagues. In May, 1925, he undertook a visit to Scotland partly on account of his failing health, but the change of air and environment did not have the desired effect. Throughout the voyages to Scotland and from Scotland his condition gave rise to some anxiety on the part of his relatives. When nearing Ceylon he was seized with a cerebral hæmorrhage and he died at Colombo on October 31, 1925.

For some years he had acted as Physician at the Saint George District Hospital and a little over a year ago became the Senior Physician.

James McLeod leaves two daughters, Mrs. Kay, wife of Dr. W. E. Kay, of Waverley, and Mrs. R. K. Clark, M.A. The sympathy of the medical profession is extended to both.

JOSEPH FRANK STRONG HAY.

THE medical profession in New Zealand has recently lost a very prominent member. Dr. Joseph Frank Strong Hay, Inspector-General of Mental Hospitals for the Dominion, sought to regain his health in July, 1924, by taking a trip to the old country. He had been ill for about a year. On his return to Wellington, his condition became worse and he died on September 5, 1925, on board the *Ruahine* a day before her arrival.

Joseph Frank Strong Hay was of Indian birth. He was born at Lucknow in 1867. He was sent to school to England and later became a student of medicine at the University of Aberdeen. He graduated at the age of twenty-three. His first professional appointment was to the position of Assistant Medical Officer at the Perth Royal Asylum. During the following six years he worked assiduously and laid the foundation for his future career as an alienist. In 1897 he left Scotland, but was not registered in New Zealand until 1899. He served as Medical Superintendent at the Ashburn Hall Private Mental Hospital until 1904. His knowledge of psychiatry and his ability as an organizer and supervisor had by this time become well recognized. A vacancy occurred in 1904 on the staff of the Department of Mental Hospitals and Joseph Frank Strong Hay was elected Deputy Inspector-General of Mental Hospitals. He filled this post with great credit to himself and benefit to the community. In 1907 on the death of Duncan McGregor he was promoted to the position of Inspector-General. For sixteen years he worked hard and admirably as head of the service and achieved a great deal. The changes that have been effected in the control and treatment of persons suffering from diseases of the mind during this period have been very far reaching. Frank Hay took full advantage of the progress of the science and art with which his life's work was associated, and the liberality of his mind became reflected throughout the mental hospitals of New Zealand. When his health began to fail, he struggled to maintain his control and to keep the work up to a high standard. The effort was

detrimental to him and he was forced to give in after a year. He was a very popular leader; his colleagues held him in very high regard and his friends loved him. He was a man of learning and had a vast store of knowledge of English literature.

Books Received.

TAYLOR'S PRACTICE OF MEDICINE, by E. P. Poulton, M.A., M.D. (Oxon), F.R.C.P. (London), with the assistance of C. Putnam Symonds, M.A., M.D. (Oxon), F.R.C.P. (London), and H. W. Barber, M.A., M.B. (Cambridge), F.R.C.P. (London); Thirteenth Edition: 1925. London: J. and A. Churchill. Royal 8vo., pp. 1079 with illustrations. Price: 28s. net.

Medical Appointments.

Dr. James Sydney Alexander Rogers (B.M.A.) has been appointed Acting Medical Superintendent of the Hospital for the Insane, Beechworth, Victoria.

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Dr. Maynard Scott Deneen (B.M.A.) has been appointed Government Medical Officer at Yerranderie, New South Wales.

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Dr. W. Bruce Fry (B.M.A.) has been appointed District Medical Officer and Vaccinator at East Kirup, Western Australia.

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Dr. Albert Stephen Furness has been appointed Second Assistant Medical Superintendent, on probation, Hospital for the Insane, Goodna, Queensland.

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Dr. Dominic Victor Sheil (B.M.A.) has been appointed Acting Government Medical Officer at Mackay, Acting Health Officer and Acting Visiting Surgeon to His Majesty's Gaol, Mackay, Queensland.

* * * *

Dr. Ernest Chenoweth (B.M.A.) has been appointed Government Medical Officer at Mackay, Health Officer and Visiting Surgeon to His Majesty's Gaol, Mackay, Queensland.

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Dr. Henry Edwin Pellew (B.M.A.) has been appointed Medical Inspector of Schools, Education Department, South Australia.

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Dr. Dorothy Sorby Adams (B.M.A.), Dr. Edith Clement (B.M.A.), Dr. Mavis Victoria Grant (B.M.A.) and Dr. Mary Courtenay Puckey (B.M.A.) have been appointed Medical Inspectors of Schools, South Australia.

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Professor Frederic Wood Jones and Dr. Richard Sanders Rogers (B.M.A.) have been appointed Members of the Board of Governors of the Public Library, Museum and Art Gallery of South Australia.

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Professor Frederic Wood Jones has been appointed an Honorary Commissioner to inquire into and report upon the methods of conducting Faunal Reserves and of marketing their products in Great Britain and the Continent of Europe.

* * * *

Dr. Reginald Francis Matters (B.M.A.) has been appointed Surgeon, Night Clinic Department, at the Adelaide Hospital.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.

AUSTIN HOSPITAL, HEIDELBERG, VICTORIA: (1) Junior Resident Medical Officer; (2) X Ray Technician; (3) Honorary Pathologist.

PUBLIC HOSPITAL, FREMANTLE: (1) Senior Resident Medical Officer; (2) Junior Resident Medical Officer.

WESTERN AUSTRALIAN PUBLIC SERVICE: Psychologist.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Ceduna, Wudinna (Central Eyre's Peninsula), Murat Bay and other West Coast of South Australia Districts.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

DEC. 8.—Tasmanian Branch, B.M.A.: Branch.
DEC. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
DEC. 9.—South Sydney Medical Association, New South Wales.
DEC. 10.—New South Wales Branch, B.M.A.: Branch (Ordinary).
DEC. 10.—Victorian Branch, B.M.A.: Council; Election of office-bearers, 1926, and appointment of Committees.
DEC. 10.—South Australian Branch, B.M.A.: Council.
DEC. 11.—Queensland Branch, B.M.A.: Branch (Annual).
DEC. 11.—Western Australian Branch, B.M.A.: Council.
DEC. 14.—New South Wales Branch, B.M.A.: Organization and Science Committee.
DEC. 15.—Tasmanian Branch, B.M.A.: Council.
DEC. 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
DEC. 16.—Western Australian Branch, B.M.A.: Branch.
DEC. 18.—Queensland Branch, B.M.A.: Council.
DEC. 19.—Eastern Districts Medical Association (Port Macquarie), New South Wales.
DEC. 31.—South Australian Branch, B.M.A.: Branch.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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